

MODERN MEDICINE FOR NURSES



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TO MY HUSBAND

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PREFACE

This new edition has been prepared in collaboration with my friend and colleague Dr. Rosemary Miller. Her nursing and medical qualifications made her help and advice doubly valuable. Much of the book has been re-written and the rest thoroughly revised. Amongst others, the sections on the treatment of heart failure, renal failure and the nursing of the unconscious patient have been re-written. There is a new chapter on Obesity and Anorexia Nervosa and a new introduction to the chapter on Children's Diseases.

Several diseases appear for the first time in this edition. Among them are a few rarities, such as phaeochromocytoma, porphyria and disseminated lupus. I include them because nurses will often see patients in whom such diagnoses are considered, and they will help with the necessary investigations. However, the emphasis throughout the book is on the common diseases and practical problems encountered in the wards of a general hospital.

When revising the chapters on Children's Diseases and Skin Diseases I consulted Dr. Henry Giles and Dr. Stanley Wood; they gave me much help and advice and I should like to thank them for their kindness.

P. A.

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PART I

GENERAL PRINCIPLES

CHAPTER ONE

REST

MOST of a nurse's working day in a medical ward is spent in enabling the patients to rest. She makes their beds, brings them their meals and attends to their toilet, so that they themselves have nothing to do except to get well. The fight with his disease leaves the seriously ill patient with no energy for any other exertion—and even such mild activity as getting dressed and undressed increases the body's work by 25–60 per cent. Hence the importance of good nursing in the treatment of all medical cases.

Rest is the basis of many medical treatments, and in some illnesses it is the only treatment we can provide. No drugs are known which will cure cases of smallpox, measles, nephritis, influenza, hepatitis or glandular fever. In these and in many other diseases all we can do is to “keep the patient in tranquillity while nature effects the cure.”* Even when curative drugs or other treatments are available, rest is still essential; the bad pneumonia case needs good nursing as well as antibiotics if he is to recover.

Complete bodily rest is often necessary to rest some particular organ. Suppose a man has pneumonia. Part of one lung is acutely inflamed. It is generally advisable to rest an inflamed part as much as possible. It is clearly impossible to immobilise the lung, but its movement is reduced to a minimum during the quiet breathing of a person at rest. Even moderate activity increases lung movements three or four times. Such increased activity of an inflamed organ tends to spread infection and delay healing. Then, again, the more the lung moves the more pain the patient must suffer. This is because in pneumonia the pleura covering the affected lung and lining the chest wall is nearly always inflamed too, and with each breath the two roughened surfaces grate over each other. Increased exertion means more work for the heart too, which must beat more quickly and with greater force. In pneumonia the heart is sometimes damaged by bacterial poisons (toxins) and death from heart failure may occur in a severe case.

*Pickles' “Epidemiology in Country Practice.”

The heart is temporarily weakened by circulating poisons in many acute fevers, which is one of the reasons for keeping the febrile patient in bed. Some bacteria produce toxins which are particularly deadly in their effect on the heart. Diphtheria toxin is such a heart poison, so diphtheria patients need prolonged and complete rest.

It is clear that the heart and lungs can only be rested by resting the whole body.

Special methods are often necessary to rest certain other organs—for instance, special diets in kidney or liver disease.

Finally, it must always be remembered that complete rest is impossible if the mind is strained and over-worked.

Mental rest depends partly on freedom from responsibility, such as the patient finds in hospital, where everything is done for him. It is increased by confidence in his treatment, in the doctors and in the nursing staff. Where there is a friendly atmosphere in the ward, when patients know that they are being well looked after and that a nurse is always at hand, then they will be at ease and free from anxiety.

Patients are sometimes tired out by their visitors. Arrangements for visiting hours are discussed in Chapter 8, but here it may be mentioned that a nurse may need all her tact in persuading visitors to leave when they are clearly exhausting the patient.

The Dangers of Prolonged Bed Rest

Just as overdoses of the most valuable drugs do harm instead of good, so one can have an overdose of rest. In a patient too long confined to bed the muscles grow flabby and bedsores tend to develop. The circulation is slowed down, particularly in the legs, where the stagnant blood may clot in the veins. Infections—sometimes fatal—may attack the lungs or the bladder. Many chronic rheumatic patients get much worse if they stay in bed too long, as joints become fixed and muscles waste. A paralytic who has had to spend a few weeks in bed, say, with bronchitis may find himself unable to get about as he used to do. Frail old people often seem to lose their urinary control when bedridden, and to become incontinent. And most nurses are familiar with the "bedridden mentality" in which inactivity is accepted and the patient loses all desire for a normal life.

The period in bed has been cut considerably compared with what was once advised. Even when bed rest is necessary, its dangers must be remembered and guarded against once they are recognised, like the risks of any other form of treatment. *Weak muscles* can be

REST

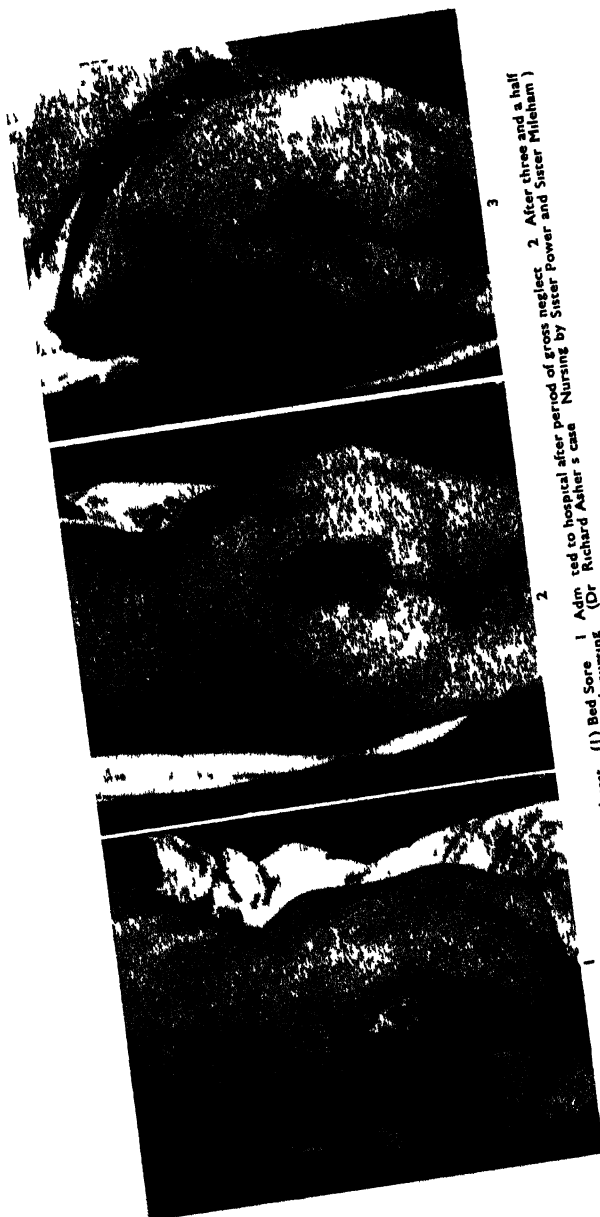


Fig 1. The dangers of prolonged bed rest (1) Bed Sore (2) After three months good nursing (3) After eight months good nursing (Dr Richard Asher's case Nursing by Sister Power and Sister Milham)

strengthened by encouraging the patient to move his limbs around in bed as he gets stronger. Massage and exercises under the direction of a physiotherapist are very valuable. I need hardly describe the prevention and treatment of *bedsores*, as techniques have been pretty well perfected in all hospitals with a high standard of nursing, and student nurses are generally drilled in such techniques from the moment they enter the wards. Bedsores are the result of the impaired circulation wherever there is undue pressure. They are more likely to occur in immobilised patients, in the very fat and the very thin and in the incontinent. They can nearly always be prevented by proper nursing—*e.g.* by changing the patient's position regularly, by the use of air cushions and by using applications which harden and dry the skin, such as spirit and dusting powder. Water repellent barrier creams (such as silicone preparations) are useful when the patient is incontinent; they prevent waterlogging of the skin and so help to prevent bedsores.

Sheets must be changed frequently if the patient is incontinent.

Urinary infections are particularly likely to occur among women patients who are in bed for any length of time. They are difficult to prevent because we do not fully understand how they arise, but it may help to give the patient plenty to drink. *Lung infections*, such as *hypostatic pneumonia*, are very common among old people who are bedridden for whatever cause—stroke, heart failure or fractured femur. If a person lies flat on his back for a long time and moves very little, all the secretions from his bronchi tend to collect at the back of the lung in a stagnant pool which readily becomes infected. The aged and infirm are seldom deep breathers, and they may be too feeble to cough this infected material up from the bottom of the chest. To prevent hypostatic pneumonia all bedridden patients should sit up as much as possible and should move around in the bed, or be moved by the nurses.

The *clotting of blood in the veins* (venous thrombosis) may also be prevented by encouraging as much activity as the patient's condition allows. If *rheumatic* or *paralysed* patients have to be kept in bed, they, too, must preserve whatever movement is still possible. If they cannot do this themselves the nurses must do it for them, putting the stiff or paralysed limbs through a regular series of movements several times a day.

Sleep

*Sleep is the best, the most complete rest we know. In deep, dreamless sleep the mind is at rest and the body's work is reduced



Fig. 2. The dangers of prolonged bed rest: (2) Contractures. There was nothing wrong with this old lady except that she had been in bed too long. (Dr. Richard Asher's case.)

to a minimum—the muscles relax, the heart beats less quickly, the blood pressure falls and breathing is slow and steady. Both mind and body need this complete relaxation, this

“Balm of hurt minds, great nature’s second course,
Chief nourisher in life’s feast.”*

Even healthy people often feel worn out after a sleepless night—though this fatigue may be due to worry rather than lack of sleep, for the sleepless are generally anxious. (See the case of insomnia—Macbeth—already quoted.) A sick man needs a good night’s sleep even more than a healthy one. During the long hours of the night the pains and discomforts of illness seem to grow, while all sorts of worries crowd into the patient’s restless mind. Such lack of rest slows—and may even prevent—recovery; in olden days it was said that “pneumonia patients sleep or die.”

To ensure sleep is therefore an important part of the treatment of any sick person. Insomnia is common in high fever, heart failure, high blood pressure, anæmia and many other conditions. It may be due to the patient’s condition, to anxiety, pain, coughing, breathlessness, itching, or any discomfort such as excessive heat or cold. Needless to say, sleep is also prevented by any disturbance in the ward.

Complete silence is hardly to be expected in a many-bedded ward. There is generally at least one patient with a cough and several who snore. The nurses have to move about, attending to those who need them, the doctor and the night sister do their rounds, perhaps an emergency is admitted in the small hours. Then, again, it is never completely dark—so no wonder the new patient finds it hard to drop off in such surroundings. Perhaps he falls asleep at one or two a.m., and it seems to him that but a moment has passed when the night nurse wakes him at 5.30 for his toilet.

In treating insomnia we must first remove, if possible, all barriers to sleep; if the patient is still sleepless he may need a sleeping draught or tablet. Aspirin or morphia may help the patient who is kept awake by pain; an itching rash calls for a cooling lotion, a cough for a dose of linctus. The nurse’s part is to discover any minor irritations which are keeping her patients awake, adjusting ventilation, pillows and bed-clothes as required. Above all, she must keep her ward quiet.

**Macbeth*, Act II, Scene 2.

Though complete silence is hard to achieve, nurses can do a great deal to help their patients to sleep by seeing that doors do not bang or windows rattle, by moving about quietly and by wearing non-squeaky shoes with rubber heels. And let the acutely ill patients get as much sleep in the morning as they can; do not wake them at five just because this is the usual routine. Ward routine should always be adapted to the patients, not the other way round.

Finally, a word about the part played by *drugs* in the treatment of sleeplessness. It is most important for all nurses to know something of the action of sleep-compelling drugs (hypnotics) because, though most of them can only be given on a doctor's prescription, in hospital it is generally the night nurse who decides if and when to give the dose. "Butobarbitone gr. 3 p.r.n." (as required) is written on the treatment card and the nurse is left to decide when it *is* required. I suggest that a hypnotic, if required at all, should be given early. It is often the practice to give the drug only when it is clear that a patient is sleepless—generally about midnight or one a.m. Now most of these drugs take about half an hour to act, so that it may be nearly two a.m. before a patient gets to sleep after a dose at 1.15. If he is then awakened at 5.30 he has not had enough sleep. If a hypnotic is ordered "p.r.n." it should be given at "lights out", at least on an acutely ill patient's first night in hospital, when few sleep well and all need to do so. If no hypnotic is ordered and the patient seems likely to need one the night nurse might well contact the doctor early in the evening and ask for a prescription. The doctor's night round is generally too late for a drug prescribed then to give the patient a good night's sleep.

Here are some of the commoner hypnotics. The *barbitone* group includes some of the most familiar—butobarbitone ("soneryl"), amylobarbitone ("amytal"), quinalbarbitone ("seconal") and pentobarbitone ("nembutal") are all widely used. Butobarbitone (dose gr. 1-3) and amylobarbitone (gr. 1½-5) take effect in about half an hour and act for 5 or 6 hours. Quinalbarbitone (gr. ½-3) and pentobarbitone (gr. 1½-3) act more rapidly and for a shorter time. Barbiturates are generally administered as tablets or capsules, but preparations for intramuscular injection are available. *Chloral* (gr. 5-30) is a very safe hypnotic; it is given as a syrup or draught and has an unpleasantly bitter taste. Its safety makes it a suitable hypnotic for children. *Paraldehyde* (½-2 drachms) is also unpleasant to take, and causes bad breath, but is safe and effective. It can also be given by injection, which is useful in cases of vomiting. Injections of paraldehyde are, however, painful. *Methyl pentynol* ("oblivon")

250 mgm., may be used when anxiety is the chief cause of wakefulness.

Hypnotics should never be withheld because of the remote danger of habit formation. A patient in hospital is in exceptional circumstances, and he is very unlikely to feel the need of the drug once he returns to health and his normal life. The treatment of *chronic insomnia* is a much more difficult problem, which cannot be considered here.

This discussion of sleep seems to have taken up a great deal of space. The subject is important because the sleepless are not resting and rest is so often the basis of medical treatment.

In the recovery of health food is second only to rest in importance, so the subject of the next chapter is Food.

CHAPTER TWO

FOOD

THE nurse's business is to see that her patients get better. To do this they need enough food of the right sort. In general, sick people require the same kind of food as the rest of us, except in acute illness and in various conditions needing special diets, which will be described later. Here then is a brief account of the kinds of food necessary to health.

A Healthy Diet

Food supplies the fuel from which the body derives the energy necessary for all life and activity. It also supplies the materials which build up a child's growing body and make good the everyday wear and tear in an adult. Finally, certain foods protect us from illness.

Food as a Fuel.—The main "fuel" is the carbohydrate in food—that is, starch and sugar. Carbohydrate supplies half the total energy value of the average British diet. Flour, oatmeal, rice and other grains, and foods made from them such as bread, puddings, pastries and porridge, are starchy foods, and so are potatoes and other root vegetables. Most of these are cheap and filling, so the poorer the family—or the country—the more bread and potatoes, or rice or yams, they are likely to eat. Sugar, jam, honey, sweets and so on are also energy-supplying foods.

Most children and many adults eat far too many sweets* and sweet things, which is bad for the teeth. Many more children's teeth are carious today than during the [redacted] war, apparently because of the quantities of sweets consumed.

Fat supplies about 38 per cent. of the energy value of an ordinary diet. Fat is a more concentrated form of fuel than carbohydrate, and, weight for weight, provides more than twice as much energy as starch or sugar. Unlike carbohydrate, fat can be stored in the body and used in emergency. A fatty meal makes the eater feel satisfied—or even over-full—because fat delays the emptying of the stomach, which may still be full of food an hour or two after a greasy meal. Milk, eggs and cheese contain fat, and so do herrings and certain other fish; butter, cream, lard, dripping, margarine and olive oil

*The British eat more sweets per head than any other nation in the world.

consist largely of fat. The last two are vegetable fats, the others, animal fats.

Animal fats often contain vitamins A and D (see below) and are valuable foods. They are expensive, and the richer the individual, or the nation, the more animal fat they usually eat. Recently, too much animal fat in the diet has been held responsible for the high rate of coronary artery disease (p. 71) among the more prosperous classes and nations. Vegetable fats, such as margarine and olive oil, are often thought to be healthier; the question is still undecided.

Food as a Body Builder.—*Proteins* are necessary for the growth of young bodies and the repair of full-grown ones; they constitute the remaining 12 per cent. of an average diet. Meat, liver, kidneys, fish, eggs, milk and cheese are rich in protein; peas, beans and lentils contain rather less, and it is called "second class protein," as opposed to the "first class protein" of animal origin, because it lacks certain important ingredients.

An adult needs about 75 grammes ($2\frac{1}{2}$ oz.) of protein a day. If he ate half a pound of cod, a good helping of peas, an ounce of cheese, an egg and a glass of milk during the course of the day, he would have had this amount. Children, however, need three or four times as much protein as adults in comparison with their size—which means that a child of six needs as much as a 10 stone man. Pregnant women also need plenty of protein to supply the needs of the fœtus (the unborn child).

"Poor" diets are usually poor in protein as well as fat. Fortunately, most people in this country today eat enough protein foods, but in many parts of the world the diet is grossly deficient in protein, leading to "hunger oedema," weakness, and poor growth and development.

Minerals in Food.—The body also needs certain mineral salts for proper growth and repair. The following are the most important:

Iron.—This is necessary for proper blood formation and *anæmia* results if there is a shortage of iron in the food. The subject is more fully discussed in the section on anæmia. Women are more apt to become anæmic than men, because of the monthly loss of blood during the menstrual period. Pregnant women need plenty of iron, and so do children. Iron is found in lean meat, liver, green vegetables, eggs, poultry and certain fish. A woman could get a day's supply of iron by eating a good helping of liver, or a mutton chop, or ten eggs, or $\frac{1}{4}$ lb. of mustard and cress or three oz.

of wrinkles. It will be seen that iron-containing foods are expensive (except perhaps the wrinkles), and poverty generally means that the diet lacks iron, causing anæmia among the women and children.

Calcium and Phosphorus.—These two elements are necessary for the development of bones and teeth; thus they are especially needed by young children and by pregnant and lactating women. Milk and cheese are rich in both calcium and phosphorus, and eggs contain plenty of phosphorus. A pint of milk and an ounce of cheese per day will supply a child or a pregnant woman with enough calcium for her special needs.

Salt (Sodium Chloride).—This is necessary to life. The average diet contains more salt than is necessary in ordinary conditions. If a person sweats a great deal, however, he loses much salt in the sweat and he may then need extra salt. People who sweat copiously and then quench their thirst with water may suffer from cramp, weakness and other symptoms of salt depletion. Fireman's cramp is a well-known condition in ships due to this combination of circumstances.

Protective Foods.—The Vitamins. Food may satisfy hunger and yet cause disease if it lacks vitamins—chemical substances which are present in minute amounts in certain foods. Gross vitamin deficiencies cause obvious well-recognised disorders, but the effects of minor deficiencies are often unnoticed. Serious vitamin deficiency is very uncommon in this country, except among aged and infirm people living alone, in alcoholism and certain diseases where food is inadequately absorbed.

Vitamin A.—The skin and membranes lining the digestive and respiratory tracts suffer if there is a shortage of this vitamin. The skin becomes dry and scaly, and the cornea may lose its transparency, so that vision suffers, and the patient becomes more susceptible to infection. *Night blindness* is another result of vitamin A deficiency. Vitamin A is found in milk, butter, carrots, green vegetables, and most plentifully in cod liver and halibut liver oil. An adult's daily requirements are contained in 6 oz. of milk, 2 teaspoonfuls of cod liver oil or a tablespoonful of spinach.

The Vitamin B Group contains several vitamins. Lack of *vitamin B 1* (thiamin, aneurin) causes a type of polyneuritis called *beriberi*, common in certain tropical countries. The *B 2* group includes *nicotinamide* and *riboflavin*. Nicotinamide prevents a tropical skin disease called *pellagra*; lack of riboflavin causes *sore lips* and *cracks*

at the corners of the mouth (cheilosis). Poor appetite, constipation, and low disease resistance have at various times been attributed to minor deficiencies of one or other of the B vitamins. Meat, liver, oatmeal, yeast extracts such as Marmite, contain these vitamins in varying proportions. A good helping of porridge and one of liver or two of meat would supply ample vitamin B for one day. Brown bread contains B vitamins. White bread is now fortified with vitamin B preparations to make up for the naturally occurring vitamins which are lost in preparing white flour.

Vitamin C prevents scurvy, a disease once common among sailors, in which bleeding from the gums and into the tissues occurs. Scurvy is still occasionally seen among bottle-fed babies. Here again we are not sure what symptoms are caused by moderate lack of vitamin C; weak muscles, slow recovery from injury, anæmia and poor resistance to infection have all been put down to shortage of vitamin C. The vitamin is found in oranges, lemons, black currants, green vegetables and potatoes. Overcooking destroys it, and greens which have been overboiled and then kept hot for a long time before serving contain practically no vitamin C. One orange, half a tomato, two or three new potatoes or a helping of cabbage would supply enough vitamin C for one day.

Vitamin D is essential for the proper formation of bones and teeth. Lack of this vitamin causes *rickets*, a disease of toddlers in which the bones are soft and deformed, and the legs tend to bend under the child's weight. Pregnant women, infants and young children need plenty of this vitamin; how much vitamin D an adult needs is uncertain. Vitamin D is found most plentifully in cod liver and halibut liver oil; milk, cream and butter contain some, but not much. Dairy produce has a higher vitamin content in summer than in winter, because sunshine enables the cow to manufacture more vitamin D. Human beings can also manufacture their own vitamin D if they are exposed to sunlight. Infants and young children should be given one teaspoonful of cod liver oil or three drops of halibut oil a day, especially during the winter, unless they are fed on milk preparations containing added vitamin D (see p. 365).

'Roughage.'—All food contains a certain amount of indigestible residue which is passed out of the body in the stools. Constipation is apt to result if the diet has too little roughage-containing foods such as green vegetables, fruit, oatmeal and brown bread.

Food which needs chewing such as apples, raw vegetables, meat and crusts is good for the teeth and gums, which, like other body

tissues, need work if they are to be healthy. In most civilised countries too much of the food is soft and pappy. This, together with the excess of sweet things already mentioned, encourages dental decay.

Water.—Last, but not least, water is necessary to life. In this country most people drink about two or three pints of fluid a day, but much more is necessary if they sweat a great deal. Remember this when nursing sweating patients.

How much food do we need? If plenty of food is available most people eat about the right amount. Some, however, eat too much, and of these some become too fat. (We do not know why *all* those who overeat do not become too fat, or not to the same extent.) Obesity should always be avoided. Apart from the appearance, many diseases—including all forms of heart disease and arthritis—are made worse by obesity. Extreme obesity shortens life, chiefly by its effect on the heart. (See Chap. 17, p. 250).

The amount of energy which the food has to supply is measured in *calories*. A working man needs 3,000–4,000 calories a day, a woman 2,700–3,000, a boy in his 'teens 3,000–3,400, a child of nine needs 2,000–2,300 calories and so on. A man lying in bed all day only needs about 1,600 calories. It will be seen that children use up more energy than adults do, in comparison with their size, and they also need more of various special foods.

That is a short account of the kind of food a healthy person needs to keep him robust and vigorous, and the same kind of food is needed by a sick person if he is to recover quickly.

Food for Hospital Patients

A patient in hospital needs less food than a man who is doing a full day's work because, as we have seen, a man lying in bed only uses up about half as much energy as a working man. But he may need *more* of some of the special ingredients of a healthy diet. For instance, in high fever the body's protein is rapidly broken down, and this protein must be replaced or the patient will waste and lose strength. Most fever patients have little appetite, and would not fancy large helpings of such protein foods as steak or kidneys and bacon; they need plenty of milk, eggs, egg custards, perhaps fish. Then again, hospital patients are likely to need more vitamins than healthy people. If vitamin C speeds up healing processes, a man with a healing gastric ulcer should have plenty of vitamin C. Vitamin A,

which is supposed to strengthen our defences against infection, should be plentifully supplied to hospital patients, who are usually exposed to all kinds of infections in the ward. They need their butter, eggs, carrots and liver even more than the rest of us.

Thus a hospital diet should be smaller in amount but better in quality than a healthy person's, which means cutting down the starchy foods such as bread and potatoes and giving more of the body building and protective foods already described. Unfortunately, this is not always done; hospital diets, instead of leading the way, have often lagged behind what is expected by an increasingly nutrition-conscious public. In many hospitals expense used to be the main consideration, and, though fantastically expensive drugs were freely prescribed, the patients' diets seldom included fresh fruit or vegetables because they are rather dear.

So much for what the average patient should eat. In most hospitals, however, the nurses have little to do with the actual catering. Their part is to serve the meals, and to see that the patients eat their food, and a most important part it is. Patients in bed often have a poor appetite, and it is the nurse's job to tempt them to eat—the most perfect diet, crammed with vitamins, does no good if it is all left on the side of the plate.

How can hospital meals be made more appetising? First of all it is most important that hot food should be served really hot, and cold dishes really cold. Too often patients are put off by the sight of tepid gravy congealing on a cold plate. With the usual gas cooker in the ward kitchen it should be possible to re-heat any of the hot food which has cooled off in transit from the main kitchen, and to heat the plates. If the ward has a refrigerator, stewed fruit, salads and other cold dishes can be served with a refreshing chill in hot weather.

It is encouraging to give the patient a small quantity of food and then to offer a second helping if he wants one. A man with a poor appetite is easily discouraged by an enormous plateful of stew, with potatoes piled high—he picks at it dejectedly and leaves most of it as it gets cold, whereas he may finish off a small, neatly served helping with relish and ask for more. If possible, serve meals on individual trays with traycloth, napkin and cruet—the meal will then look much more tempting. (Shortage of staff and of linen may make such methods difficult at times.)

Serving a patient with an appetising meal not only gives him pleasure but improves his digestion. If a man sits down to dinner with a good appetite it means that that dinner will be digested all the

quicker by a well-primed stomach. Experiments have shown that, just as the mouth waters at the thought, sight or smell of good food, so does the stomach—a flow of gastric juice occurs so that the digestion of this appetising food begins the moment it enters the stomach. On the other hand, anything unpleasant slows down digestion in the stomach and may stop it altogether, when the patient is sick.

Hospital staffs—doctors, nurses, cooks, caterers and dieticians—have a great responsibility in seeing that the patients get the food they need in order to get better. But only the ward nursing staff can see that the patients eat the food provided, and can assist digestion by serving the food in an appetising way.

RECORD KEEPING

(a) THE TEMPERATURE CHART

NURSES, like doctors, are concerned with diagnosis as well as treatment. Their part in diagnosis is to carry out certain investigations of which the most important are taking the temperature, pulse and respiration rates and testing the urine. These four investigations are part of the routine examination of any patient, no less and often more important than the doctor's examination of heart, lungs and abdomen.

As the clinical thermometer is almost the first instrument the student nurse learns to use, let us start with the temperature chart.

A person with a raised temperature is hardly ever well, so that the thermometer is a most useful instrument in the detection of illness. There is a complicated and beautifully adjusted mechanism which normally keeps the body temperature between about 97° and 98·6°F. however hot or cold the outside air may be. If the thermometer shows that the body is much hotter or colder than normal, we know that something is amiss. Occasionally an excitable type, burning with emotion, may show a rise of temperature of a degree or two, and violent exercise in the hot sun may have the same effect, but as a rule a raised temperature means illness.

Of course, a normal temperature does not prove that all is well. A man may be disabled by the pain of a gastric ulcer or an attack of migraine, bedridden with arthritis or dying of a cerebral tumour—all with a normal temperature.

By far the commonest cause of a raised temperature—fever—is *infection*. When harmful organisms invade the body their toxins seem to upset the mechanism which regulates the temperature, so that the blood becomes hotter. It is thought that this may help the body to overcome the invasion by speeding up the defences against infection, though it is difficult to prove this point. We do know, however, that in certain overwhelming and rapidly fatal infections the patient's temperature may be normal throughout.

Apart from infection, fever often occurs after a stroke, and in various other conditions where the brain is damaged. A rise of temperature usually occurs after a coronary occlusion. Burns and fractures, even if uninfected, may also cause fever, and so may

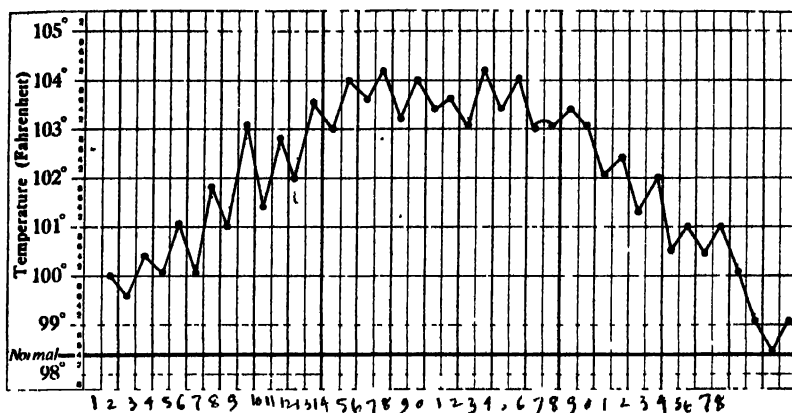


Fig. 3. Temperature chart of a case of Typhoid Fever.

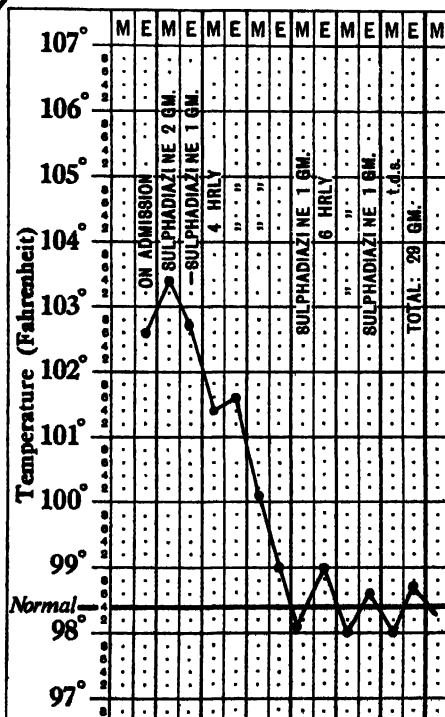


Fig. 4. Temperature Chart of a case of Lobar Pneumonia, successfully treated with sulphadiazine.

injections of vaccines or serum. Quite a number of drugs, including the sulphonamides, may cause fever in certain people, and sometimes it is difficult to know if the temperature is due to the disease or to our treatment.

A single temperature reading may tell us that the patient is ill; the temperature chart often tells us much more. In some diseases the chart is so characteristic that it suggests the diagnosis. The "stepping stone" rise followed by sustained fever which subsides after two or three weeks is typical of *typhoid fever*. In *lobar pneumonia* there is generally a sudden rise to 101° or more, and the temperature stays at this level until the patient is given the proper sulphonamide or antibiotic treatment. (Inadequate treatment may lead to a slow, wavering fall of temperature. In untreated, uncomplicated pneumonia the high temperature is maintained for 7-12 days, when it falls rapidly—the crisis of the disease—but this once familiar chart is rarely seen today.) A high, swinging temperature with daily spikes often means an infection in the blood stream—*septicæmia*. In the tropics the *malarial* temperature chart is a familiar sight, showing peaks every one, two or three days, according to the type of infection.

Thus the temperature chart helps in the diagnosis of disease; it is still more useful in the early detection of complications. For instance, suppose a man with lobar pneumonia has had a normal temperature for a week; one evening the thermometer reading is 100° , and next morning it is higher still. At once we must look for some complication, of which perhaps the most likely is *empyema* (pus in the pleura). An acute infection of one sort or another may complicate any illness. Women are specially apt to develop *urinary infections* when confined to bed for whatever cause, and the only sign of such infection may be a rise of temperature. Young children may suddenly shoot alarmingly high temperatures of which the cause may be most difficult to trace, though perhaps the *ears* are the most frequent culprits in such cases.

In chronic febrile (feverish) illnesses the temperature is often an important indication of the patient's progress. For example, in pulmonary tuberculosis persistent fever shows that the disease is still active; sometimes it is an indication for fresh treatment—e.g. with steroids (p. 118).

The Normal Temperature

If we can understand how the temperature is controlled in the healthy person, it will help us to understand both the symptoms and

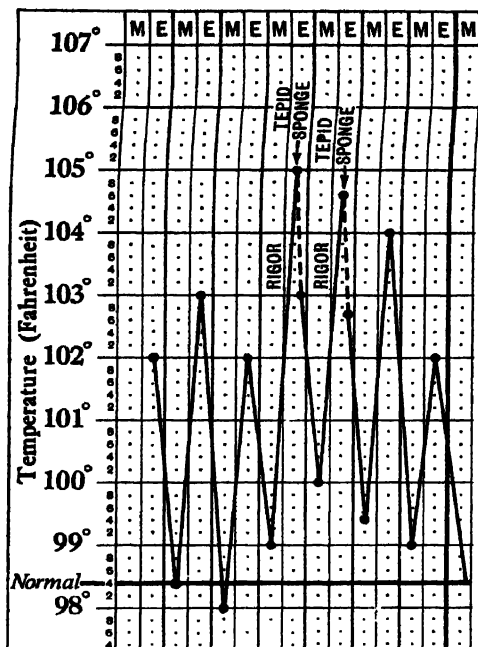


Fig. 5. Temperature Chart of a case of septicæmia.

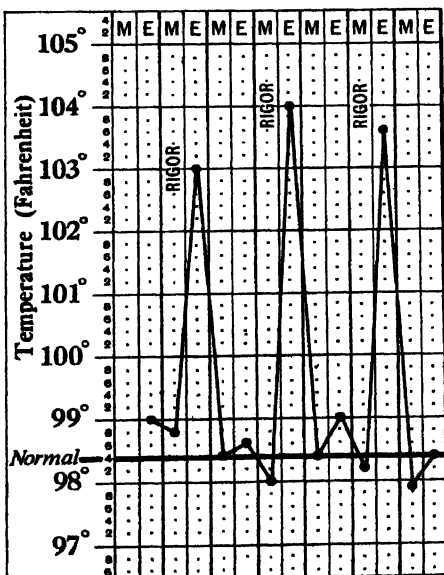


Fig. 6. Temperature Chart of a case of Malaris (Benign Tertian).

treatment of fever. This control is normally so efficient that a man's temperature may still be 98.4° when the outside temperature is hot enough to grill a beefsteak (250° F.). Such a man would be red in the face and dripping with sweat. Sweat cools the body as it dries just as the evaporation of the water in a butter cooler cools the butter. The redness of the skin means that it is full of blood, and this diversion of the blood to the surface of the body helps to keep it cool. On the other hand, if you have to sit in damp clothes in a chilly waiting room exactly the opposite occurs—the body preserves its heat by diverting the blood *away* from the skin, which feels cold to the touch, and dry, because of the absence of sweat. *Shivering* also helps to keep the body temperature from falling in such cases. as the violent muscular activity produces heat.

In *fever* this same shivering, dry skin and diversion of the blood away from the surface occurs even though the patient is in a warm room. The result is a rise of body temperature. If the shivering is very violent the temperature may shoot up four or five degrees in less than an hour; such an attack is called a *rigor*. A *fall of temperature* occurs when the patient sweats and the skin flushes; at this stage he often feels much better.

The Treatment of Fever

It is usually unnecessary to "bring down the temperature" artificially because, as we have seen, moderate fever does no harm and may even do good. If, however, a patient's temperature rises above 104° or 105° the blood may be hot enough to damage the tissues. On these occasions we may have to "bring down the temperature." First let us be sure that the patient is not overheated by too many bedclothes. A *tepid sponge* may then be considered. Sponging the patient cools him just as an outbreak of sweat would do, and the temperature may fall through two or three degrees as the moisture dries. Large doses of *aspirin* also lower the temperature in fever, but such doses are apt to be dangerous in high fever and unnecessary in other cases.

Of course, the best way to bring down the temperature is to cure the disease that is causing the fever, as we do when we treat a case of pneumonia successfully with antibiotics.

Low Temperatures

An abnormally low or *subnormal* temperature is much less common than fever. It may occur during convalescence after fever, in surgical

shock, hæmorrhage, certain prostrating infections, in cold injury of the newborn (p. 359) and in a few glandular disorders such as myxœdema and pituitary failure. In certain rare cases of these dis-

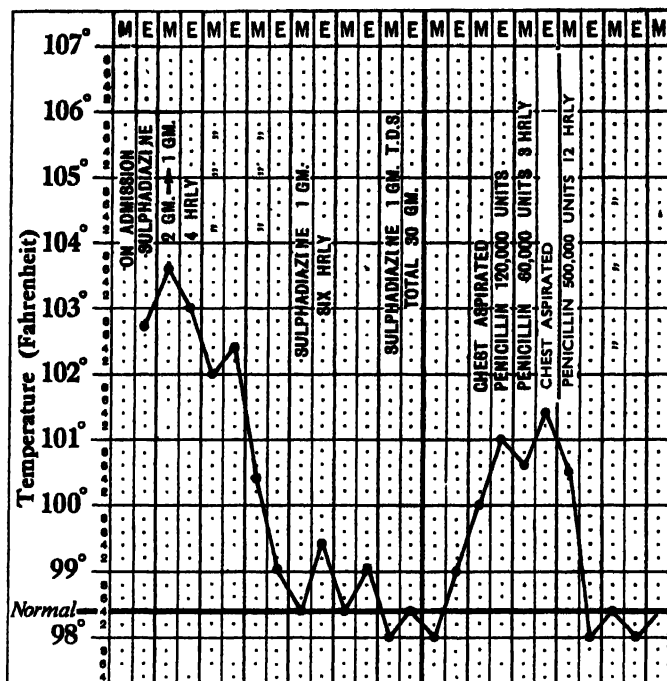


Fig. 7. Temperature Chart of a case of Lobar Pneumonia complicated by Empyema.

orders the temperature may be so low—perhaps only 85° F.—that it can only be recorded on a special thermometer (see p. 225). But by far the commonest cause of a reading of 95° or 96° is failure to leave in the thermometer long enough.

The Temperature Chart

To conclude this chapter here are a few practical points concerning the temperature chart. The temperature is generally entered twice a day; four-hourly records are required when the temperature varies from hour to hour, as in some acute infections. A four-hourly chart may be necessary in other cases, because, though in most people the temperature is at its highest at about 6 p.m., the time of the usual evening round, in some cases the peak may be at

some other time of day, and would be missed on a morning and evening chart.

If the temperature is taken anywhere but in the mouth the fact should be noted on the chart. With the thermometer in the groin or axilla the reading will be about a degree *lower* than the mouth temperature, whereas the rectal temperature is about a degree *higher*. The groin or axilla may be used if the patient cannot keep the thermo-

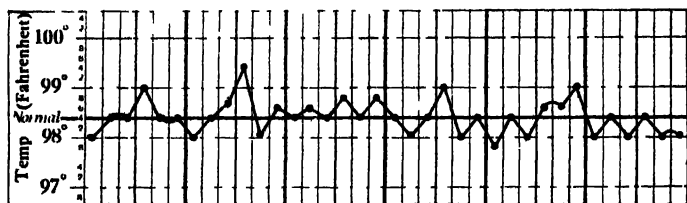


Fig 8 Four-hourly Temperature Chart of a case of Pulmonary Tuberculosis (Note that a "Morning and Evening" Chart would have shown no raised temperature in this case)

meter in his mouth, as in unconscious patients and small children. The temperature so taken, however, is often inaccurate, even if the thermometer is left in place for several minutes. Rectal temperatures are commonly taken in infants.

The temperature chart becomes a most helpful document if events such as rigors, and the most important items of treatment such as the administration of drugs or serum, tepid sponging, etc., are entered on the chart, as in the examples here given. It all helps to make the patient's condition clear to whomever is in charge of the case, and often saves much bewildered whisking over of notes. Of course, the pulse and respiration rates, bowel action and, perhaps, fluid intake and output will also be entered and are as important as the temperature; these will be the subjects of the following chapters.

CHAPTER FOUR

RECORD KEEPING

(b) PULSE AND RESPIRATION RATES

ALTHOUGH a whole chapter was devoted to the temperature chart, perhaps the importance of the pulse and respiration rates should be even more carefully stressed, as they are so often neglected. This may be because they are seldom taken without the temperature, whereas the thermometer is often used without the wrist watch. Suppose there is a question of sending a nurse off sick. If her temperature is found to be 101° she will at once be bundled off to bed, whereas a pulse rate of 115 with a normal temperature might simply be due to nervousness.

The pulse and respiration rates are very much more affected by exercise and emotion than is the temperature. So records will not be very useful unless the patient is at rest.

The Pulse Rate

Besides the normal quickening of the pulse caused by excitement or exercise, rapid beating of the heart, and hence a rapid pulse, occurs in many diseases. Particularly is this so in four groups of disorders—infections, loss of blood, heart disease and, much more rarely, disorders of the ductless glands (endocrines).

In almost any form of *fever* the pulse rate is raised, because the action of the heart is speeded up by the heat to the extent of about ten beats per minute for each degree rise of temperature. The poisons produced by the organisms responsible for the fever may also act directly on the heart, causing rapid beating (tachycardia) out of all proportion to the fever. (In typhoid and influenza the pulse is apt to be rather slow.) When the heart muscle is thus poisoned it contracts more feebly. There is a nervous mechanism controlling the circulation which keeps the amount of blood pumped out by the heart per minute more or less constant. This means that the enfeebled heart will automatically beat more quickly as less blood is discharged per beat.

All bacterial toxins may thus poison the heart, but some are more apt to do so than others. In the later stages of *typhoid fever* the pulse is often rapid and feeble, and a quick pulse may last well into con-

valescence. In severe cases of *pneumonia* the heart is nearly always affected. Not only is the pulse rapid, it is often irregular as well, due to the occurrence of *auricular fibrillation* (see below). In all such cases the pulse, rather than the temperature, indicates the gravity of the patient's condition.

The pulse rate is still more important in certain surgical conditions. For instance, in a suspected case of appendicitis the other signs may be doubtful; if a half-hourly pulse chart shows a steady rise it may decide the question of operation. Here again the tachycardia is due to the toxic action on the heart—in this case caused by the bacteria which are attacking the peritoneum around the appendix.

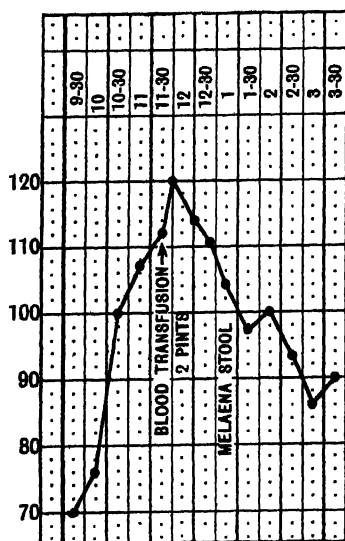


Fig. 9. Half-hourly pulse chart in a case of Duodenal Ulcer with severe hæmorrhage.

The hourly or half-hourly pulse chart is also useful in cases of suspected internal bleeding. If a patient has a bleeding duodenal ulcer the blood shed in the duodenum will eventually reach the lower bowel, causing *melæna* (black stools). But this may take several hours, and meanwhile the pulse rate will be rising, which may assist early diagnosis. (The pulse is soft in such cases and the blood pressure low; a falling blood pressure is a more reliable sign than a rising pulse rate when hæmorrhage is suspected.)

Severe loss of blood (hæmorrhage) always quickens the heart rate.

This is because less blood is reaching the heart from the veins. The poorly filled heart beats feebly, and, as when it is weakened by toxins, nervous reflexes bring about a corresponding increase in rate.

In *surgical shock* the whole circulation is enfeebled by loss of fluid, of which hæmorrhage is not the only cause. For instance, in a severe case of burns enormous quantities of fluid pour into the blisters and from the burnt areas; this fluid comes from the blood, which becomes sticky and reduced in volume. Hence the rapid, feeble pulse of the shocked patient.

As the enfeebled heart tends to beat quickly tachycardia is to be expected in many forms of heart disease. A child with rheumatic

fever whose pulse-rate is still high after the temperature has subsided has most likely got a damaged heart. The rheumatic infection attacks the heart itself, often leaving it permanently injured, unlike the temporary effect of pneumonia and other infections.

In *heart failure*, whatever the cause, the pulse rate is raised. The failing heart beats feebly but rapidly just as it does when it is weakened by bacterial toxins. After a coronary occlusion, however, the pulse rate may be low.

Finally, the heart is influenced by several of the ductless (endocrine) glands, and disorder of these glands may affect its action. The *thyroid gland* is a heart accelerator, and in *Graves's disease*, where the gland is overactive, the pulse is abnormally rapid; *myxædema*—a disease in which the thyroid may cease to function altogether—causes a *slow* pulse.

An abnormally slow pulse, like a subnormal temperature, is much less common than the reverse. It may occur in old age, starvation, among trained athletes, in cases of raised intracranial pressure, in a rare condition called *heart block* and in digitalis overdosage. The last is the most important cause of a slow pulse to remember, because so many patients are treated with digitalis, and because the effects of overdosage may be serious, though they are entirely preventable. Nurses should always report the fact if the pulse rate of a patient who is taking digitalis falls below 60.

What else can we learn about a patient by feeling his pulse? Not only the rate, but the force, volume and rhythm may give us valuable information. The pulse which beats feebly against the fingers laid on the patient's wrist tells us that the heart, too, is beating feebly, as in severe infections, hæmorrhage, shock and heart failure. In the severest of such cases the pulse can scarcely be felt. In cases of *high blood pressure* the pulse may feel unusually forcible—but beware of trying to guess the blood pressure by feeling the pulse. Even the best clinicians cannot do it. Then there is a condition called *aortic redurgitation* where a grave valve defect allows blood which has been pumped out of the heart to flow back again after each beat. This backward flow partially empties the arteries between heart beats, so that each pulsation at the wrist feels like a sharp tap followed by complete collapse of the artery. This is known as the *collapsing pulse*. It is best felt when the patient's arm is raised above heart level, as this accentuates the collapse of the artery.

The *rhythm* of the pulse is often as important as the rate and force. Normally the heart beats with almost complete regularity, save for a slight quickening during inspiration and slowing during expiration.

This alternate quickening and slowing with the breathing is called *sinus arrhythmia*. It is much more obvious in children than in grown-ups, but it is never a sign of disease.

Most people's hearts occasionally miss a beat or give an extra contraction. These extra beats are called *extrasystoles* or *premature contractions*. A premature contraction appears at the wrist as a weak beat following closely on a normal pulsation and succeeded by a pause. Premature contractions are not usually a sign of heart disease, unless they are very frequent. They are common among heavy smokers and heavy tea drinkers.

A much more important irregularity is *auricular fibrillation*. This will be more fully described in the chapter on heart failure. Its presence nearly always means that the heart is severely damaged. In auricular fibrillation the heart beats with an absolute disregard for good order and discipline. Weak beats and strong, long pauses and runs of rapid beats follow each other without rhyme or reason. A totally irregular pulse of this sort is nearly always due to auricular fibrillation.

So much for the examination of the pulse. The pulse rate is usually entered on the temperature chart as a figure, but it is often helpful to chart it in the same way as the temperature is charted, using red ink or pencil to distinguish the two lines. The results of treatment in heart cases and in others where the pulse is affected can thus be seen at a glance.

The Respiration Rate

Last but not least—though generally considered the Cinderella of the three sisters “T., P. & R.”—the respiration rate. One seldom finds that as much attention is paid to the respiration rate as to the temperature or the pulse rate. Sometimes, however, it is the most important of the three. Suppose we are examining a patient who, we suspect, may have pneumonia. His temperature and pulse rate may be high—but so they are in many other infections. But if his respiration rate is over 30 he has very likely got pneumonia. Moreover, the higher the respiration rate the more serious his condition, which does not always apply to the temperature. A child with pneumonia may easily have a temperature of 104° or over without being very ill, for children run high temperatures very easily. But the child with pneumonia whose respiration rate is over 60 is gravely ill.

In pneumonia the respiration is rapid and *shallow*. This is because the inflamed lung is less elastic than normal and will not expand fully. The patient, unable to take deep breaths, takes many shallow ones.

The pain of his pleurisy also makes for shallow breathing, and this pain often causes a little catch at the end of each inspiration—a very characteristic sign in pneumonia.

In *heart failure* the patient's respiration is also rapid and shallow. This is because the lungs are engorged with blood and inelastic. Here also the respiration rate is a measure of the severity of the patient's illness.

In *diabetic coma* the patient's breathing is rapid and *deep*. The rapid breathing is due to the accumulation in the blood of certain abnormal chemicals which act on the nerve centre which controls respiration. Breathing is deep as well as rapid because the lungs are normal. Rapid deep *hissing* respiration may be a sign of uræmia (p. 197); here too, the cause is the accumulation of abnormal chemicals in the blood.

Rapid deep breathing also occurs after a sudden severe hæmorrhage, the patient gasping for air as if he were drowning—"air hunger". A sudden quickening of the breathing may be the first sign to draw the nurse's attention to a patient who has had an internal hæmorrhage—for instance, in typhoid or duodenal ulcer.

Rapid deep breathing in an apparently healthy patient may be a hysterical symptom. This is an exaggeration of the quickening of the breathing which occurs in normal people at moments of crisis. Hysterical overbreathing can cause tetany (p. 236); this is because so much carbon dioxide is washed out of the blood that the blood becomes more alkaline; alkalosis is one of the causes of tetany.

Unusually *slow* breathing may occur in cases of brain tumour and allied conditions. This, and the associated slow pulse rate, is caused by the abnormal pressure on the centres in the brain which control these vital functions. Sometimes the breathing waxes and wanes in such a case, both in rate and depth—*Cheyne-Stokes* respiration. This type of breathing may also occur in uræmia, stroke and in the final stages of many other diseases.

Thus, careful observation of the patient's respiration will teach the nurse a great deal about her patient's condition, especially in such illnesses as those mentioned above.

CHAPTER FIVE

URINE TESTING

IN testing the urine the nurse is making another important contribution towards the diagnosis of her patient's condition, and the results of her investigations may also help to assess the patient's progress and act as a guide to treatment.

A summary of the tests in common use is given below. The rest of the chapter will describe what we can learn from these tests.

(1) *Albumin*. Filter if urine is cloudy. If alkaline to litmus, add 10 per cent. acetic acid till blue litmus paper turns red. Boil the top of a test-tube full of urine. If a *white cloud* appears add 2 drops acetic acid. If the white cloud persists albumin is present.

(2) *Sugar* (glucose). Place 4 drops of urine in test-tube. Add 2.5 ml. (about $\frac{1}{2}$ inch) Benedict's reagent. Boil for two minutes. If glucose is present a yellow or brick-red precipitate appears.

(3) *Acetone*. (a) *Rothera's test*. Take 10 ml. urine; add ammonium sulphate crystals till no more will dissolve. Add 3 drops of fresh sodium nitroprusside solution and 2 ml. strong ammonia solution. If *even a trace of acetone* is present a deep purple colour appears. (b) *Ferric chloride test*. Add 10 per cent. ferric chloride solution drop by drop until a precipitate forms and then disappears. A brownish red colour shows that acetone is present in considerable quantities.

Rapid methods of testing for albumin, sugar and acetone are also available; special tablets or impregnated paper strips are used and the colour changes compared with the chart provided.

(4) *Blood and pus*. To 1 inch of urine in a test-tube add 2 drops of tincture of guaiac. A white precipitate appears. Add 1 inch of ozonic ether. A *blue ring* at the line of junction of the solutions indicates that *blood* is present; a *green ring, pus*.

(5) *Bile salts*. Sprinkle flowers of sulphur on urine. If they sink, bile salts are present.

(6) *Bile pigments*. Filter urine. Place a small drop of yellow nitric acid on the filter paper; a play of colours indicates the presence of bile pigments.

(7) *Salt* (sodium chloride). Place 10 drops of urine in a test-tube with a pipette. *Rinse pipette*. Add one drop of 20 per cent. potassium chromate. *Rinse pipette*. Add silver nitrate solution drop by drop; shake test-tube each time. The colour suddenly turns yellow brown. The number of drops of silver nitrate solution necessary to produce this colour equals the number of grammes of salt in a litre of urine.

(8) *Phenyl Pyruvic Acid*. Add 5 per cent. ferric chloride solution to urine until a definite colour appears; a blue-green colour may indicate phenyl pyruvic acid (see below).

Of these tests the most important are those for albumin and sugar. They form part of the routine medical examination of all hospital patients, and of candidates for life insurance and recruits to the forces.

Tests for albumin

Albuminuria occurs in many acute fevers, in heart failure (p. 49), in diseases of the kidney, in pyelitis and cystitis (p. 186) and in many other conditions. It also occurs in some healthy people. Remember, also, that any non-catheter specimen may contain albumin, especially if the patient is a woman. This is because vaginal secretions, which may contain albumin, often contaminate non-catheter specimens.

Albuminuria may be explained as follows. The kidney acts as a filter, conserving substances which are of value to the body and getting rid of waste products. Albumin, a protein, is a necessary constituent of the plasma—the fluid part of the blood. If the kidney is damaged, like a leaky filter it allows some of this albumin to escape.

The kidney is temporarily damaged by toxins in acute fevers, and by lack of oxygen in heart failure—hence the albuminuria so common in these conditions. More serious is the albuminuria in nephritis, for here the kidney damage may be permanent. Nephritis may follow scarlet fever or tonsillitis, so urine tests may be required when patients are convalescent from these diseases.

Another disease which may affect the kidney is toxæmia of pregnancy. Albuminuria is one of the signs of this serious disease, so a specimen of urine is tested for albumin at each ante-natal examination.

Albuminuria occurs in pyelitis and cystitis, though the kidney may be healthy, because pus is present in the urine. Pus always contains albumin and other proteins. The pus comes from the inflamed lining of the kidney outlet or the bladder.

Tests for sugar

If sugar is found in the urine the patient may have diabetes mellitus (see p. 237). This disease often starts insidiously; the patient may have no idea that he has diabetes when the nurse finds sugar in a routine specimen. A fellow-student once found a patient's pre-operative specimen loaded with sugar. The patient turned out to have diabetes, so her operation, which was not urgent, was postponed until her diabetes had been controlled. Anæsthetics are

dangerous to the untreated diabetic, and this patient might have become seriously ill had her diabetes remained undiagnosed. This shows the importance of routine urine tests.

Diabetes is not the only cause of glycosuria (sugar in the urine) though it is the most important one. The subject is discussed on p. 238.

Tests for acetone

If the urine of a diabetic contains acetone as well as sugar it means that the patient has diabetic ketosis, which may lead on to coma. If the ferric chloride test is positive, the urine contains a considerable amount of acetone, and the patient's condition may be serious.

Acetone also occurs (without sugar) in many acute fevers in childhood, especially where there is much vomiting. It is a sign that the body's metabolism is upset, and that fat is being used up rather than sugar.

Tests for blood and pus

Large quantities of *blood in the urine* are unmistakable. Moderate amounts give the urine a smoky brown appearance. Small quantities may be found by means of the test described above, or—much more reliably—with the microscope. Hæmaturia (blood in the urine) may occur if any part of the urinary tract is injured, inflamed, or affected by a tumour. Blood in the urine is a most important sign of kidney damage in nephritis (p. 190), sulphonamide overdosage (p. 97), and bacterial endocarditis (p. 85). Anti-coagulants (p. 76) may also cause bleeding from the kidney as from other sites.

Pus is found in the urine in pyelitis and cystitis (p. 186). Large quantities form a white sediment when a specimen is allowed to stand; the sediment has a "ropey" appearance when poured from one glass to another. The microscope is necessary to find small numbers of *pus cells*.

Tests for bile salts and pigments

These tests are positive when the flow of bile is obstructed—e.g. by gallstones; they are also positive in hepatitis and other diseases of the liver.

Salt

Salt is normally present in the urine. It may be absent after excessive sweating or vomiting, for salt is usually lost in sweat and vomit. The urine in diabetic coma (p. 247) is usually salt free. When salt reappears following treatment—e.g. after a saline drip—this is a good sign.

Tests for phenyl pyruvic acid

This substance is present in the urine in a very rare disease called phenylketonuria, which, if untreated, causes mental deficiency. The importance of the test lies in the fact that it enables the diagnosis to be made in early infancy, when the baby seems quite normal. If given a special diet from this time, *the baby may develop normally*. Because of this, routine testing of all babies' urine is being carried out in some areas. Because of the difficulty in obtaining samples of a baby's urine the test is often done on a wet napkin with "phenistix," which turns green if phenyl pyruvic acid is present.

Specific gravity

As well as these chemical tests a measure of the specific gravity is done. A *urinometer* is floated in a glass of urine; the deeper it sinks the lower the specific gravity of the urine. A concentrated urine has a high specific gravity. Normally the specific gravity varies between about 1002 and 1025 (that of water is 1000). When the kidney fails, as in chronic nephritis, the specific gravity is often almost constant at about 1010, for the kidney has lost the power of producing more concentrated or more dilute urine according to the needs of the body. When the urine is loaded with sugar the specific gravity is *raised* (e.g. to 1030 or more).

Lastly, it is often important to know *how much* urine is passed, and here an "intake and output" chart is valuable, recording the total volume of fluid drunk and the volume of urine passed in twenty-four hours. (In severe diarrhoea and vomiting the volume of stools and vomits must be included in the fluid output.)

A patient who passes no urine may have *retention* or *suppression of urine*. *Retention of urine* means that the bladder cannot empty itself; the full bladder can be felt rising into the abdomen and urine can be obtained with a catheter. Retention may occur when the nervous control of the bladder is upset, as after a stroke (p. 282), or when there is a blockage such as an enlarged prostate; impacted faeces, by pressing on the bladder outlet can cause retention. In such cases the urine sooner or later begins to leak out of the bladder (*retention with overflow*). Always feel for a distended bladder in cases of incontinence.

Suppression of urine means that the kidneys have ceased to function; no urine is produced and the catheter finds an empty bladder. This is known as *acute renal failure* (p. 197). The patient rarely produces no urine at all, but he may pass only a few ounces of urine a day, and this is a much more serious sign than any amount

of albumin or blood in the urine; an increased flow of urine is hailed with relief, for it probably means that the patient has turned the corner. In *heart failure*, too, the daily output may fall below a pint a

day, instead of the normal two or three pints, because the kidney is temporarily damaged by the poor circulation. The intake and output chart then shows the results of our treatment, for, in successful cases, enormous accumulations of fluid may be passed in a day or two—15 pints in 24 hours is not uncommon. In *diabetic coma* (p. 247) the urine is often scanty because of the extreme dehydration.

Suppression of urine may occur during sulphonamide treatment if the patient does not drink enough. Certain sulphonamides tend to clog the kidney unless the patient drinks plenty of water, and so flushes the kidney with plenty of urine. If too little water is drunk the urine may become scanty and bloodstained. So give these patients plenty to drink. An intake and output chart may be required.

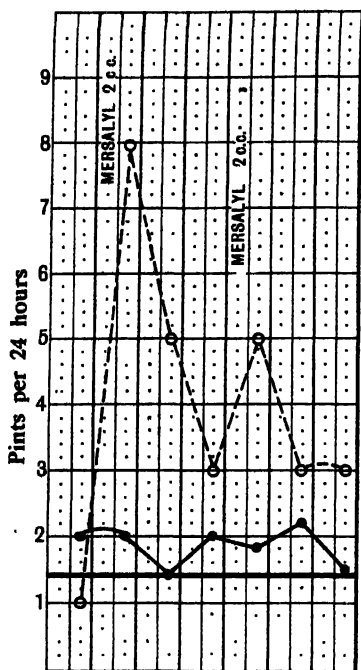


Fig. 10. Daily Fluid Intake and Output Chart from a case of Cardiac Dropsy treated with Mersalyl. Upper line output, lower line intake.

Finally, to make these all-important fluid charts clearly legible, NEVER use Roman figures when entering the volume of urine. They are difficult to read, easy to mistake (for few are absolutely certain that IX means 9 and not 11) and practically impossible to add up at the end of the day.

CHAPTER SIX

BOWEL ACTION

IN these chapters we have so far discussed the temperature, pulse and respiration rates and the testing of urine. Now we come to the last item which is usually entered on the patient's chart - the action of his bowels. In most hospitals bowel irregularity is dealt with by the nursing staff. So it is important for nurses to understand how the bowel functions in health and disease, and how to treat irregularities, for this is one of the few fields of medicine in which they are entirely responsible both for diagnosis and treatment.

The basis of medical treatment is, as we have seen, to make the patient comfortable and to help his own powers of recovery by improving his general condition. We have seen how rest and good food help him to overcome his illness. If his bowels act normally one more obstacle to recovery will be removed, and he will also feel more comfortable.

The normal adult passes, as a rule, one semi-solid motion a day. The passage of many fluid stools (diarrhœa) occurs in various disorders and will not be discussed here. The trouble that nurses more often have to treat is *constipation*.

Our aim is to see that the patient's bowels act normally during his illness, just as a normal, healthy diet is what the recovering patient needs. Now, a daily action produced as a result of a daily purge is *not* normal, so, before deciding how to treat our constipated patients, let us see how the bowels act in health.

Regular evacuation of the bowels is largely the result of habit training in childhood. The desire to open the bowels (defæcate) occurs when the pressure inside the rectum reaches a certain level. In an infant defæcation occurs automatically at this point, but habit training means that it must be postponed until the proper moment. If this training is successful the child should get the habit of having his bowels open once a day. Defæcation is partly the result of involuntary contractions of the bowel wall, partly of the "bearing down" movements of the abdominal muscles and diaphragm. If, because of indolence or lack of opportunity a deaf ear is turned to the call to stool, the rectum adjusts itself to its increased contents and the desire to empty it passes off; if this happens frequently constipation is the result, and hard, dry fæces, which can

hardly be passed even with great effort, become impacted in the rectum.

There are other causes of constipation. The food may not contain enough insoluble residue (roughage) to give bulk to the fæces; the colon may be of the absorptive type which greedily takes up more than its usual quota of water from its contents, leaving only a dry little residue to be passed on to the rectum. Then, again, proper evacuation of the bowels may be prevented by weakness of the abdominal muscles, or by the pain of piles or anal fissure.

It follows from all this that the regularity of bowel action in a hospital patient may be upset in several ways. First of all, habit

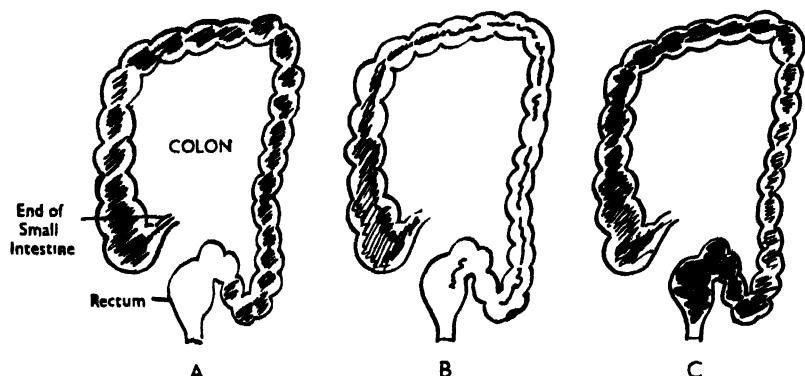


Fig 11 Diagrams of the Large Intestine

Fluid fæces enter colon from small intestine. The first half of the colon absorbs water, the second half stores the fæces. The rectum is normally empty until just before defæcation

"Greedy Colon"
Too much water is absorbed in the first half of the colon, so that the fæces are dry and scanty—one of the common causes of constipation

"Lazy Rectum"
The rectum which is emptied regularly becomes stuffed with rock-like fæces—another common cause of constipation

training is largely a matter of association and, when his entire routine is unfamiliar, the most "regular" patient may fail to produce a motion for the first few days. Then, many people find it difficult to use a bedpan, as the abdominal muscles do not come fully into play when sitting up in bed with the legs straight out. These muscles, too, are often weakened by long confinement to bed, and incapable of strong contractions. And finally, invalid diets are often lacking in the kind of foods which give bulk to the fæces—salads and green vegetables, porridge, brown bread, and so on.

So, for several reasons, patients confined to bed are often constipated. There are now two questions to be answered: does the patient need any medicine (or pills or an enema), and if so, what? First, as to the necessity of artificial purgation—it is a mistake to think that dire results will follow if no bowel action occurs for a day

or two. The vast majority of the population are firmly convinced that constipation is followed by wholesale release of deadly poisons into the system, with vague but alarming results. For this belief there is absolutely no scientific foundation. Constipation often causes some discomfort, but this is due to the distension of the overloaded rectum, not to the absorption of imaginary toxins. Since, then, the patient is not going to be "poisoned" if he fails to empty his rectum daily it is always worth waiting for a day or two to see what he can do unaided before rushing to the pill box or the enema syringe. Furthermore, there are some people who habitually pass a motion only every two or three days, and as long as this motion is not hard and dry, but is of normal consistency, these people are *not* constipated, nor should they be treated as such.

To overcome the bedpan difficulty, if the patient is not very ill he should be allowed out of bed for his daily action, in which case he will often be successful, even when in bed he appeared to be completely constipated. (A bedpan placed on a chair may be tried if the patient cannot go to the toilet and no commode is available.) In order that there should be plenty of faeces of the right consistency in the bowel the diet should include fresh and dried fruit, cooked and raw vegetables, unless his condition makes such food inadvisable; there should be brown bread as well as white and plenty of water to drink.

In spite of all these precautions patients will often be in genuine need of assistance in the evacuation of their bowels. Either they are habitually constipated and have developed the purge habit, or genuine and obstinate constipation results from their illness. Or the slightest straining at stool may be dangerous, as in some heart disease, or constipation might aggravate some local condition such as piles.

In patients who have difficulty in passing hard, dry motions a lubricant such as liquid paraffin may be sufficient. The dose is about one tablespoonful three times a day. It has no irritating action on the bowel and so is quite safe to use. Where there are piles it often eases the passage of hard, stiff faeces over the sore place.

Then there are substances such as agar and vegetable mucilage, which both lubricate and moisten the faeces, as they tend to draw water into the bowel. A flaky preparation of agar may be sprinkled on stewed fruit in teaspoonful doses. One to four teaspoonfuls of vegetable mucilage (e.g. "isogel") are soaked in a glass of water, and the resultant jelly-like fluid is swallowed. Any of these harmless lubricants may be combined with laxative drugs when they are ineffective alone, such as compound emulsion of liquid paraffin (paraffin, agar and phenol phthalein).

When these preparations fail to act one of the vegetable laxatives may be tried. These act on the lower bowel, chiefly the colon. They increase its peristaltic movements and hurry the bowel contents along to the rectum. They are apt to cause griping and colic, so an antispasmodic such as belladonna is often given at the same time. The most useful varieties are cascara and senna; podophyllin and aloes are too drastic for general use. The dose should be such that one motion of normal consistency results—several fluid motions means that an overdose has been given. *Senna pods* are useful, as the dose can be graded for each patient; five to twelve pods soaked in water for twelve hours and the infusion drunk overnight should have the desired effect next morning. *Cascara* as pills (2–8 gr.) or liquid extract (30–60 minims) is another useful drug. It is combined with senna and an anti-spasmodic in Mist. Cascara Co. and with rhubarb in “Veg. Lax.” The popular Beecham’s pills contain aloes and rhubarb. Phenol phthalein (1–5 gr.) is a synthetic substance rather similar in action to the vegetable laxatives. All these drugs, being slowly acting, should be given overnight.

The *saline aperients* are very popular and in small doses are generally harmless, but they tend to produce a watery stool, and should not be used in cases of serious illness and collapse where this loss of fluid would be dangerous. They act on the whole bowel, unlike the vegetable group whose action is on the colon, by drawing water into it. They are quickly acting, and a teaspoonful or two in water on waking should result in a motion after breakfast. Large doses should not be given. Aperients of this class are Epsom salts (magnesium sulphate), Glauber’s (sodium sulphate), the tartrates and citrates, Eno’s (tartaric acid and sodium bicarbonate) and Kruschen’s salts (a mixture of Epsom and other salts).

The main trouble with all these laxatives is that they act by hurrying the motions through the small intestine or colon, whereas the stoppage is often in the rectum alone. As Chevalier wrote of purges in 1819: “The whole intestine is teased and pained for the defective action of that part of it which is most remote from their influence.” If the peristalsis of the upper part of the intestine is sufficiently violent its contents may be driven on and may eventually succeed in forcing a passage, when the patient will pass a few rock-like masses followed by a gush of fluid fæces. But sometimes violent purges, which are always undesirable, are not even effective. In obstinate rectal constipation it is better to give a small enema or suppository. The commonest suppository consists largely of glycerine, which absorbs water when inserted into the bowel, thus

lubricating and softening the fæces. It is always worth trying, for it may supply just that stimulus the sluggish rectum needs, together with a lubricant which enables it to empty itself. The patient is then spared the discomfort of an enema, which is quite an ordeal to many. Regular enemata given every three days or so are necessary in many paralysed patients who have not full control of their bowels. They may also be advisable in severe heart cases, high blood pressure and stroke, where straining at stool is to be avoided at all costs. But in most cases the enema syringe is used in an emergency rather than as a routine.

Artificial methods of emptying the bowel should never become a daily routine. Once tided over the first few days in hospital, a patient may well be able to carry on without assistance, and he should always be given the chance of doing so. Finally—though this should hardly need saying—no aperient should ever be given to anyone, old or young, until the need for it is obvious. It is still the practice in some institutions to give a purge to each patient on admission without waiting to see what he can do on his own. And in many medical textbooks one comes across the phrase (generally in an account of a disease about whose treatment little is known), “The patient is put to bed and given a brisk purge.” Why, one asks, why? Suppose he was just going to have his bowels open—why should he have to empty further tracts of his intestine of incompletely digested, fluid fæces? Constipation need not be sought where it does not exist, and when present, it should be treated with the minimum of interference and the maximum encouragement to the patient to empty his bowels regularly without medical aid.

THE CONTROL OF CROSS INFECTION

THE previous chapters have dealt with the nurse's part in the diagnosis and treatment of disease. Now we come to an aspect of her work which has come much to the fore recently; the control of cross infection. By cross infection is meant the infection of one patient by another while in hospital. Patients can also be infected in hospital by the doctors, nurses and domestics. Here are some examples of ward infections.

(1) A girl with a rheumatic heart developed tonsillitis while in a general medical ward. The infection, as often happens in such cases, was followed by a flare-up of her rheumatism. It was then found that a patient at the other end of the ward had a chronic running ear. The same strain of streptococcus (the particular germ concerned) was isolated from both cases.

(2) A nurse came on duty on an infants' ward with a cold. Within three days several babies had an upper respiratory infection and one developed pneumonia.

(3) In a large measles ward in a fever hospital some of the children had running ears and sore throats. The beds were well spaced and scrupulous barrier nursing precautions were observed (see below). The kindly ward cleaner, however, as she swept round the beds, would pick up fallen toys from the floor and replace them on the cots. Among 20 patients in this ward arose four fresh cases of running ear, which proved in each case to be due to an infection acquired from another patient.

(4) A nurse with a boil started work in the nursery of a maternity unit. Three of the babies developed septic skin conditions. In each case the strain of staphylococcus was the same as that recovered from the nurse's boil.

In each of those wards patients acquired a new illness while in hospital. It is with the prevention of such illnesses that we are concerned.

Infectious diseases are those caused by *germs* or organisms (bacteria, viruses and so on). They may be acquired by healthy people from those already infected either directly or indirectly. Apart from the common infectious fevers, which are usually treated in special

hospitals, there are many conditions caused by organisms which will be found in general medical wards. Such are pneumonia, meningitis, influenza, cellulitis, tonsillitis, running ears, impetigo and the common cold, to give a haphazard selection. Many of these conditions can be passed on from patient to patient or from staff to patient, unless constant care and vigilance are maintained.

Methods of infection.—The commonest routes by which organisms gain access to the body are the respiratory tract, the digestive tract and the skin—that is, they may be inhaled, swallowed or picked up. All these methods of infection may occur in the ward. Patients may

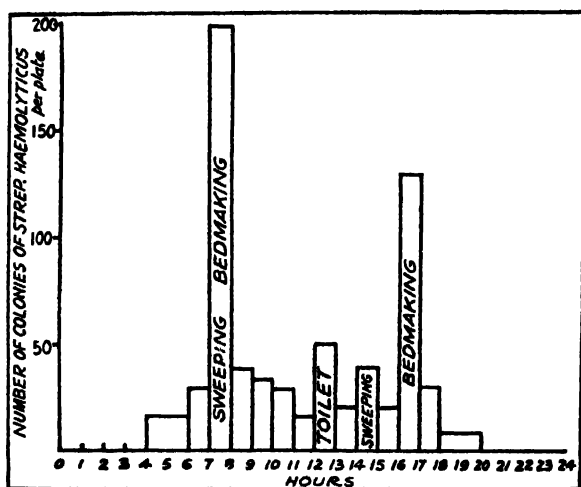


Fig. 12. When are there most germs in the air of the ward? Plates of bacterial culture medium were exposed in the ward at different times of day. Many more bacteria could be cultured from the plates exposed during sweeping and bedmaking.

inhale, swallow or pick up organisms coming from other patients or staff. How do these germs pass from one person to another?

In the first of our four examples the infection was probably carried by DUST, though the doctors' or nurses' hands may have been responsible. Mrs. Brown had said nothing about her running ear as she had had it all her life. She kept it plugged with a small piece of cotton wool which sometimes fell on the floor when removed. Dried pus and organisms then mingled with the dust. At 7.30 each morning the floor was swept and particles of germ-laden dust whirled in their teeming millions in all directions. Miss Smith inhaled some of this dust, developed tonsillitis and a recurrence of acute rheumatism.

The *nurse with a cold* coughed and sneezed out droplets of moisture laden with germs. Infants have little resistance to the virus of the common cold, which in them may cause a serious illness.

The *kindly cleaner* in the measles ward could not help picking up some of the bacteria from the toys she obligingly retrieved from the floor. These organisms she then transferred to other toys. Small children invariably put their toys into their mouths, from which the germs only had to travel a short distance to the ears they infected.

The *nurse with a boil* probably carried staphylococci both *on her hands* and *in her nostrils*, for in any staphylococcal skin infection most of the skin and the nose are, as a rule, heavily contaminated. Thus she could infect the babies while handling them or breathing over them.

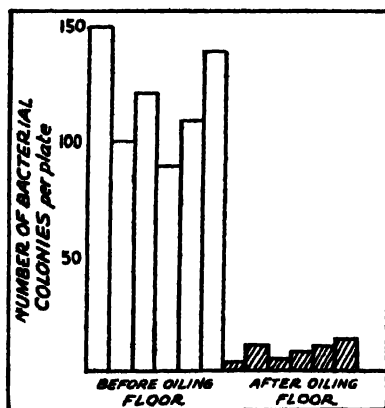


Fig 13. Oiling the floor reduces the number of germs in ward air even during sweeping

Here we have examples of four methods of cross infection against which we must be constantly on our guard. How can such infections be prevented?

Dust, that most efficient carrier of infection, must be kept down at all cost. It can be literally "kept down" by treating the floor with *spindle oil*. This oil makes the dust slightly sticky, so that it can be swept up into matted rolls, instead of flying about in the air in the bad old way. The daily floor sweepings of one ward contained 100,000,000 live bacteria. Fig. 12* shows how the bacterial population of the air increases during sweeping and bedmaking, when

*From the Medical Research Council War Memorandum "The Control of Cross Infection".

millions of germs are stirred up and given the chance to find another victim. Fig. 13* shows how spindle oil reduces the sweeping danger. *Bedmaking* spreads bacteria because of the infected fluff which is shaken out of the blankets. Nearly a million virulent organisms were found in the blankets of a case of running ear. Treating the blankets with "technical white oil" reduces the scattering of fluff. Blankets made of cellular cotton or towelling are now used in some hospitals; unlike wool blankets they can be boiled, and in addition, they scatter less fluff during bedmaking. *Damp dusting* is better than the use of a dry duster, which flicks thousands of organisms off the shelves and lockers. All crockery, food and instruments must, of course, be protected from dust. And, finally, nurses should not dust and sweep; it makes them more likely to act as carriers of disease.

Droplet infection can be prevented only by the isolation of the infected case. Nurses with heavy colds should go off duty. Often a mistaken zeal prevents this. Dr. Bevington, in "Nursing Life and Discipline,"† says: "In the course of this inquiry I interviewed nurses on duty suffering from such heavy colds as would have entailed their being sent home from any modern office, if only in order to protect the rest of the staff. It seems curious that hospital standards should, in this respect, fall below those of sound commercial practice." If shortage of staff makes it impossible to send victims of colds off duty, they should at least be kept off children's, maternity and surgical wards and sent where they can do least harm. They should in all cases wear efficient masks. These must be discarded on removal, not tucked into an apron pocket for further use.

Unfortunately, healthy nurses often carry virulent germs in nose or throat. Moreover, these germs are usually resistant to penicillin, and often to other antibiotics, so may cause infection in patients which cannot be cured with the common antibiotics. When both patients and staff harbour antibiotic resistant organisms, as often happens, only a high standard of nursing technique, with wide spacing of beds and plenty of ventilation, will prevent an outbreak of ward infections. Under-staffed, over-crowded wards invite infection, however good the nursing.

Infection carried on the hands, clothes and other articles, as in our third example, is prevented by the strict observance of *barrier nursing* technique.

*From the Medical Research Council War Memorandum "The Control of Cross Infection".

†H. K. Lewis, 1942.

The aim of barrier nursing is to stop bacteria being carried on the hands and clothes of nurses (or doctors or domestics) from one patient to another. Infectious cases—e.g. babies with gastro-enteritis, patients with septic wounds—are barrier nursed, whether they are in cubicles or in an open ward; if in an open ward the beds must be widely spaced. There must be a plentiful supply of water for handwashing by each bed, and also of gowns for the doctors, nurses and domestics. No one may touch the patient or any of his belongings (bedding, clothes, dressings, crockery, etc.) without first putting on a gown. Before leaving the cubicle (or the bedside), the nurse scrubs her hands and forearms, dries them on a clean towel, discards the towel, and removes the gown. (Hands are washed *before* removing the gown to prevent contamination of the *inside* of the gown, and hence of the clothes of the next wearer.)

Soap containing a disinfectant (such as chlorhexidine) helps to disinfect the skin of the hands, and a disinfectant cream or lotion, applied after washing, is also valuable.

The barrier-nursed patient must have his own supply of crockery, cutlery, etc. which must not come into contact with those of other patients.

Staphylococcal nasal carriers among hospital staff represent a problem as yet unsolved. Sometimes 80 per cent. of all nurses prove to be nasal carriers of staphylococci which are nearly always resistant to penicillin, and often to other antibiotics too. Creams containing an antibiotic to which the organisms are sensitive smeared inside the nostrils twice a day for ten days may markedly reduce the carrier rate, but relapses are common. Also, there is the danger of encouraging the development of resistance to yet another antibiotic.

Nurses should always report septic skin conditions such as boils, for it has been shown that the organisms from such lesions are apt to be more virulent than those from healthy carriers.

In *bowel infections* such as typhoid the bacteria concerned leave the body in the faeces and, sometimes, in the urine, so that the most scrupulous attention must be paid to the disposal of these excreta. Otherwise a few germs may be accidentally picked up and transferred to another patient, who may then develop the disease. A milder and a much commoner form of bowel infection which is prevalent in many institutions these days is epidemic diarrhoea—a mild form of dysentery, and one of the few infections to increase during recent years. In such conditions the hands become infected after using the toilet and carry the organisms elsewhere. Hands should always be washed on leaving the W.C.

Foot-operated W.C. cisterns are much more hygienic than those operated by the usual plug or lever, which is contaminated before the user has had a chance to wash his hands. The door-handle will also be contaminated unless there is a wash basin (preferably foot-operated) *inside* the lavatory.

Patients using bedpans must also have facilities for handwashing. Proper facilities for sterilising bedpans are essential.

The prevention of *wound infection* is vital in surgical wards. Wounds may be infected by airborne organisms or by those carried on doctors' or nurses' hands. In medical wards such infection may attack the skin incision when long-running transfusions or intravenous drips are given. Thrombosis may then occur in the vein; even a fatal septicæmia is not unknown. To prevent such infection scrupulous asepsis is necessary when setting up the drip and when changing the dressings. Plentiful sterile cotton wool dressings should be used to keep airborne bacteria out of the wound.

In this chapter we have only touched the fringe of the huge subject of the control of cross infection. The reduction of these infections involves every department of a large hospital and every rank of the hierarchy. Hospital architecture and equipment, catering and laundry arrangements, medical and nursing organisation, nursing treatment and ward cleaning must all play their part, but ultimately the protection of the individual patient depends on the ward nursing staff. Conscientious barrier nursing is a most laborious and time-consuming process; the observing of dust-laying routines or the wearing of masks may seem tiresome and unnecessary. But the neglect of these precautions can result in catastrophes, and it is better, though less dramatic, to keep one's cold from a heart failure case by going off duty than successfully to nurse him through the pneumonia he might otherwise develop.

CHAPTER EIGHT

GENERAL WARD MANAGEMENT

THE best run ward is the one in which patients make the speediest recovery. In such a ward the medical and nursing treatment of the patient's physical conditions will be of the highest standard, while the general atmosphere will encourage in him a spirit of confidence, cheerfulness and the desire to get well. The mental side of recovery is particularly important in a medical ward, especially among long-stay patients. Such patients easily succumb to boredom, depression and lassitude, or to restlessness and worry. Any such state of mind impedes recovery. The quick turnover, the constant coming and going and the drama of a surgical ward generally makes for a more optimistic atmosphere. This chapter contains a few notes on the maintenance of cheerfulness among medical patients.

I am assuming, of course, that the medical and nursing treatment are such as to inspire the patients' complete confidence. Such confidence is the basis of success, especially in the treatment of chronic illnesses, as has been known from the days of witch doctors onwards. If the doctors, nurses and domestics are cheerful and friendly as well as efficient the patient will be all the more encouraged.

Everyone is influenced by his surroundings. Modern hospital architects are fully aware of this, and more and more effort is being made to make new wards pleasant to look at as well as easy to run. Unfortunately many hospitals still have to do the best they can with grim nineteenth-century wards of many beds and few amenities. In most general hospitals it will be found that any modern wards are on the surgical side, whereas the worst of the 30-bedded horrors are considered good enough for the "chronics". This is grossly unfair, as the comfort of a ward that houses patients for months or even years is surely more important than that in which they only stay for two or three weeks.

Still more effort, therefore, is needed to maintain a cheerful atmosphere if one is unfortunate enough to work in a dreary, old-fashioned ward. *Flowers*, of course, make a great difference. Any flowers brought in by patients' friends should be arranged to the best advantage, and supplemented during the winter by evergreens, berries, catkins and buds. Unless the patient objects to the smell, his flowers need not be removed at night. Plants are supposed to use up

oxygen and to give off carbon dioxide, but as a large bunch of daffodils gives off less carbon dioxide during the whole night than does a man in nine or ten breaths, the effect is entirely unimportant.

Light and air are important both to health and comfort. We may be more concerned to see that there is brisk enough circulation of air to sweep away bacteria and to reduce the risk of cross infection, but what the patient chiefly notices is the presence or absence of draughts. A judicious arrangement of screens may prevent draughts even when plenty of windows are open.

The ward routine should, as far as possible, be adapted to the patients' health and comfort rather than to the doctors' convenience or the spotless appearance of the ward. The reason given for the unnaturally early hour of reveillé in many hospitals is that a later start would mean that the ward would not be ready in time for the doctor's round. It would be better to let the patients sleep on a little, even if the doctor should occasionally see a crumpled counterpane or an unwashed face.*

Patients are usually greatly cheered though they may also be overtired by their visitors. If the visiting hours are on two afternoons a week a patient often has to go seven days without a visitor, as his friends may not be free on a week-day. At the end of two hours' solid talk on the Sunday he is then exhausted. Most hospitals now have a daily visiting half-hour between 6 and 7 p.m. This is found to suit the patients, who get frequent but not tiringly lengthy visits, the visitors, who come straight on from work, and the nurses, who finish with the washing and bedmaking before the visitors arrive.

Once on the road to recovery, all patients need something to occupy their minds and hands. Some people are content to read for seven or eight hours a day and, given a supply of books, are never at a loss for something to do. Women patients nearly always have their knitting or sewing and, at a pinch, are generally glad to mend or mark hospital linen rather than do nothing. Men are much harder to occupy. Much has been learnt of the value of giving patients something interesting and constructive to do during convalescence. This has now been dignified by the term Occupational Therapy (therapy = treatment), thus indicating that its purpose is to help the patient to get well, not solely to keep him happy. Large military convalescent hospitals, orthopædic, mental and tuberculosis in-

*See "The Pattern of the In-Patient's Day" (H.M.S.O.)—a Central Health Services Council report published since this book was written—for a most valuable discussion of this problem.

stitutions usually employ full-time occupational therapists. Nurses who work in hospitals without an occupational therapy department have to use great ingenuity in finding something for their patients to do. The difference some occupation makes in a ward is amazing. In one male T.B. ward, full of disgruntled, bored patients, a craze for knitting was started by a nurse who taught one man to knit. The fashion spread until most of the men had sweaters or scarves under construction. The atmosphere at once changed to one of cheerfulness and optimism. Rugmaking, toymaking, leather work and painting can all be done in bed, if relatives can be persuaded to supply materials. All these jobs are messy and may therefore be frowned on by lovers of the spotlessly white, smoothly stretched counterpane. Here is another occasion where the patient's welfare must take precedence over the spit and polish of the ward.

Only a very few of the ways in which a patient's recovery may be speeded have been mentioned. Many more will suggest themselves to anyone of experience and imagination. If one remembers that, in most cases, what is best for comfort is best for health, and, without exception, what increases confidence and cheerfulness advances recovery the rest soon follows. Only the nurses, because of their day in, day out contact with the patients are in a position to supply those stimuli to recovery which are lacking in the most potent drugs. The patient fully realises the nurse's absolute pre-eminence in the treatment of illness, as is shown by the conversation of anyone recently discharged from hospital and by the fact that, for every letter received from a grateful patient by the doctor, the average nurse or sister gets about a dozen.

below) or *purulent* if a respiratory infection is also present—as it so often is. *Wheezing* is the result of spasm of the bronchi.

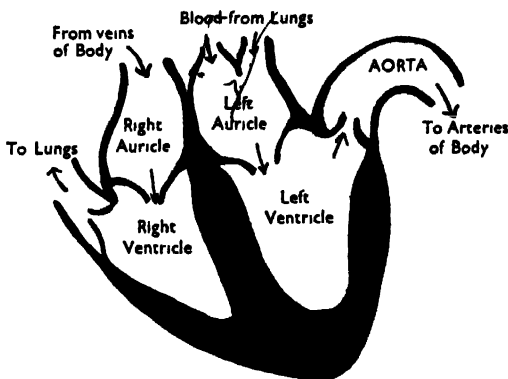


Fig 14 Diagram of the Heart

By studying the direction of the flow of blood it can be seen that, if the right side of the heart fails blood will accumulate in the veins of the whole body causing generalised congestion of veins and oedema. If the left side of the heart fails blood will accumulate in the lungs causing pulmonary congestion and oedema.

Oedema is caused partly by stagnation of the blood in the *capillaries*, partly by the inefficiency of the *kidneys*. Fluid leaks from the congested capillaries both because of the increased pressure and because the walls are damaged by lack of oxygen. The kidneys are inefficient because of the poor circulation; they produce little urine, and the urine contains albumin—a common sign of kidney damage. Water and salt—which are also excreted by the normal kidney—accumulate in the body resulting in oedema. The oedema fluid accumulates in the lowest parts of the body—the legs, if the patient is up and about, or the back, genitals and back of the thighs if the patient is in bed.*

Cyanosis is also due to capillary stagnation, because the blood remains so long in the capillaries that it loses more of its oxygen than normally, and so becomes darker in colour. Cyanosis may also be due to imperfect oxygenation of the blood, as in pulmonary oedema.

The *veins of the neck* are engorged like the rest of the venous system and so stand out prominently.

The *pulse* is rapid and feeble in severe cases, the rate rising, as usual, as the heart beat weakens (p. 23). In many cases the pulse is also totally irregular. Runs of quick beats, long pauses, weak beats and strong follow each other without rhyme or reason. The ventricles are beating with the same complete irregularity, and some of the

*Fluid may also accumulate in the pleural cavity (pleural effusion, hydrothorax) and perityneum (ascites).

beats are so feeble that no pulsation is felt at the wrist, so that the heart rate may be faster than the pulse rate. A heart rate of 160 with a pulse of 120 is quite common in such cases. This condition is called *auricular fibrillation*. In these cases the apical rate should be recorded as well as the pulse rate.

When the heart is beating normally the auricles contract first, and this contraction sends an impulse down a special strand of muscle

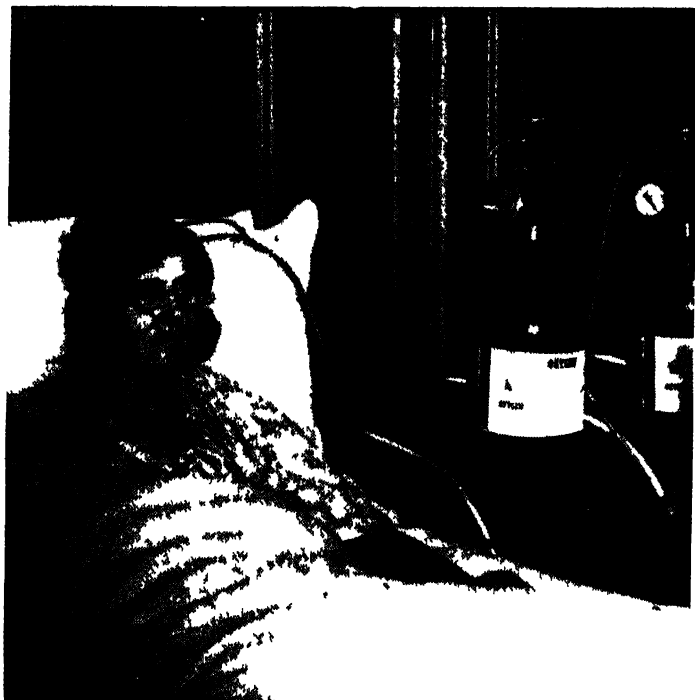


Fig 15 Oxygen administration double nasal catheter method

called the *bundle of His*, which triggers off the contraction of the ventricles one tenth of a second later. In auricular fibrillation there is no normal contraction of the auricles; instead, the whole auricular wall *filibrates*, minute waves of contraction flickering now here, now there across its surface, some 300-400 per minute. Hundreds of irregular impulses pass down the bundle of His, bombarding the ventricles with stimuli to contract. They are incapable of responding to all of them, but they may achieve a rate of 150-200, combined with complete irregularity of pace and volume.

The other symptoms mentioned above may also be attributed to

the stagnation of blood and the poor oxygen supply in the tissues—the *irritability* to lack of oxygen in the brain, the *loss of appetite* and *nausea* to congestion of the stomach. *Insomnia* is usually the result of breathlessness and cough.

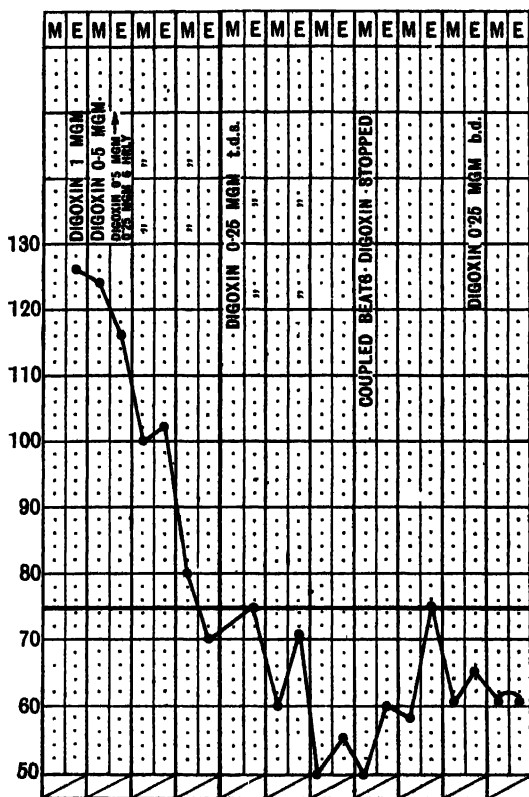


Fig. 16. Pulse Chart of a case of Auricular Fibrillation treated with digitalis.

The Treatment of Congestive Heart Failure

If the cause is known (see below) and is treatable—e.g. thyrotoxicosis, anaemia, bacterial endocarditis or respiratory infection—the *cause must be treated*; rapid improvement of the heart action often follows. Respiratory infection so often precipitates heart failure, even when other causes of heart disease are present, that a course of antibiotics is given in very many cases.

The treatment of heart failure follows from our knowledge of the workings of the failing heart. These are the most important parts of the treatment: rest, posture, diet and salt restriction, oxygen, digitalis,

aminophylline and diuretics. Ascites and hydrothorax may require drainage. Venesection (bleeding) is occasionally recommended.

(1) *Rest*.—This is the first necessity—rest for the body, rest for the mind, rest for the heart should be as complete as possible. The patient should be disturbed as little as possible, and all nursing procedures carried out with the utmost gentleness and skill. Any symptom which disturbs his rest should be treated—for instance, a patient with a cough may need a linctus. If he finds it difficult and uncomfortable to use a bedpan, a commode is often better for him—it has been shown that balancing on a bedpan increases the work of the heart more than being moved on to a bedside commode. If the patient is constipated, a mild aperient or a small enema may be necessary. Straining at stool is to be avoided.

A heart failure patient often needs no sedative, and *none should be given unless ordered by the doctor*. If the patient is very restless, chloral or a barbiturate may be prescribed. Morphia relieves severe breathlessness and distress, *but is dangerous if there is much chest infection*—it depresses respiration and can kill the patient. In such cases barbiturates, even, may be dangerous; paraldehyde in small doses may then be the safest sedative to give.

(2) *Posture*.—The patient is propped up with a back-rest and many pillows; or an adjustable cardiac bed is used, if available. The patient may feel most comfortable leaning forward on to a bed-table. Some patients prefer to be nursed in an armchair. As already described, the diaphragm moves more freely when the patient sits upright, and breathlessness is relieved.

(3) *Diet*.—*Salt must be restricted* as far as possible. This is to reduce oedema. Oedema fluid contains salt, and salt in the diet helps the body to retain fluid. No salt should be added at table, and obviously salty soups, stews and fish should be forbidden. A *salt-free diet* is sometimes advised. All food has then to be specially cooked, including bread.

The heart failure patient has little appetite, and normally eats little for the first few days. Later, small, nourishing, appetising meals should be given. The patient should drink *between* meals to avoid overloading the stomach. (If the stomach is distended by bulky meals and big drinks, the heart, which lies just above, is displaced and its action embarrassed.) If the patient is too fat a reducing diet will be necessary.

(4) *Oxygen*.—This is often necessary when the patient is very blue and breathless. It is given by means of a polythene or rubber mask or double nasal catheter; an oxygen tent may also be used, but

greatly complicates nursing. Oxygen has to be used with great caution in *cor pulmonale* (see p. 78).

(5) *Digitalis*.—This remarkable drug, widely used in the treatment of all types of heart disease, is extracted from foxglove leaves. Its action was first described by the great eighteenth century Birmingham physician, William Withering, who took the trouble to investigate an old wives' tale that "foxglove tea" was good for the dropsy.

Digitalis has two main actions: it improves the *force of contraction of the failing heart*, and it *controls the heart rate* in auricular fibrillation. *Digitalis* does not affect the abnormal action of the auricles; it damps down the conduction of impulses along the bundle of His. The auricles fibrillate away as fast as ever, but the ventricles receive fewer stimuli and beat at a slower rate; they have time to recover between beats and to fill more completely and thus beat more strongly and efficiently. More blood is discharged per beat, less remains in the overfilled venous system, and the symptoms resulting from venous congestion are relieved. The patient breathes more freely as pulmonary congestion decreases; oedema lessens because the kidneys, no longer engorged with blood, produce a normal amount of urine, and also because there is less stagnation of blood in the tissues. (As we have seen, the *digitalis* was first used as a cure for dropsy. But it is effective only in oedema due to heart disease, acting as it does on the heart and only indirectly on the kidney.)

Digitalis is always given in large doses first, so as to build up an adequate concentration of the drug in the body; this is called *digitalisation*. Smaller maintenance doses are then given and *must often be continued for life*. Digoxin is usually given at first, as it is more rapidly absorbed than *digitalis folia* (*digitalis* leaf). A first dose of 1–2 mgm. of digoxin is often given, and in an hour striking improvement may be noticed. 0.25 or 0.5 mgm. doses eight hourly are then given until the maximum benefit is obtained, after which a maintenance dose of 0.25 mgm. digoxin, or 1 gr. *digitalis folia*, once or twice a day will usually be required. In auricular fibrillation our aim is to bring the resting pulse rate down to about 70 per minute.

Toxic Effects of Digitalis.—*Digitalis* irritates the stomach, and produces nausea and vomiting, if too large a dose is given. Heart failure patients often feel sick at first, but if nausea and vomiting start after some days of treatment, they are more likely to be due to *digitalis* overdosage. Overdosage may also cause cardiac irregularities—e.g. *coupling of beats* (pairs of beats alternating with long pauses) or *excessive slowing of the pulse*. This is because the damping down of conduction along the bundle of His has gone too far, producing partial *heart block*. If any of these toxic signs are noted, *report them to the doctor immediately*. Unfortunately we do not always know if a patient has been having *digitalis* before admission, so that symptoms of overdosage may appear even after moderate dosage. Toxic symptoms are more likely to appear if the patient is also receiving chlorothiazide or mercurial diuretics (see below).

(6) *Aminophylline*.—This drug is also extremely useful in heart failure. It increases the rate and depth of respiration. It relieves spasm of the bronchi (e.g. in bronchitis, asthma or cardiac asthma—see below). It probably has a direct effect on the heart too, and increases its output. An intravenous injection of aminophylline (250–500 mgm. in 10–20 ml. solution) is often given at the start of the treatment of heart failure; the effect may be dramatic, especially with wheezy patients. Thereafter the drug may be given by mouth (one or two 100 mgm. tablets three times a day). Unfortunately large doses by mouth are apt to make the patient sick. *Aminophylline suppositories* are useful in such cases; a suppository last thing at night may ensure a restful night for the wheezy patient.

(7) *Diuretics*.—These drugs act on the kidney, increase the flow of urine and so relieve oedema. We have already seen that digitalis relieves oedema, among its other effects. In severely oedematous patients, however, diuretics are always given as well.

(a) *Mercurial diuretics* (e.g. mersalyl, “neptal”). They are usually given by intramuscular injection once or twice a week. The dose is 2 ml.

(b) *Chlorothiazide* (“saluric”) has the advantage of being given by mouth. The dose is 1–2 gm. daily, and it is given for four or five days out of seven, to prevent overdosage. It is a most effective drug. Mercurial diuretics may be given at the same time. Other oral diuretics are also available.

Toxic Effects.—Chlorothiazide increases the output of certain salts (*electrolytes*) from the body fluid—the loss of salt is, in fact, an essential part of its action as a diuretic. The main salts lost are those of *sodium* and *potassium*. (Ordinary table salt is sodium chloride). However, if the body loses too much sodium or potassium there may be serious symptoms. Chlorothiazide is apt to cause a severe loss of potassium. ~~This may cause~~ symptoms such as listlessness, weakness and irritability, and the response to the diuretic is poor. Because of this, some form of potassium is usually given with chlorothiazide (e.g. potassium chloride 1 gm. t.d.s., or potassium citrate mixture, $\frac{1}{2}$ oz. t.d.s.) Fruit juice contains potassium and is a useful addition to the diet.

Diuretic treatment is controlled by (a) clinical observation, (b) fluid input and output charts (see p. 31) and (c) estimation of the potassium in the blood. The nurse is often the first line of defence against overdosage on all these counts. (a) She may be the first to notice symptoms such as irritability. (b) She keeps the input-output charts. These are of first-rate importance and must be accurately filled in. It is useless to ask a confused patient how many cups of tea he has had: the volume drunk must be recorded *at the time*. (c) She may be the first to see the reports on the serum potassium, and must at once report to the doctor if the level is below normal (3·8–5·1 milli equivalents per litre, or m.Eq./l.)

(8) *Other Forms of Treatment.*—It may be necessary to drain the fluid from the abdomen or pleural cavity in ascites or hydrothorax, if the volume is so great as to increase breathlessness by compressing the lungs and displacing the heart. (In some cases the effusions clear up in the course of treatment in the same way as the oedema fluid.)

Venesection used to be a common form of treatment in congestive heart failure, and is still occasionally used. About a pint of blood is withdrawn, usually from one of the arm veins, by means of a large-bore needle. The pressure of blood coming into the right side of the heart may also be reduced by applying tourniquets to the limbs, just tight enough to prevent or slow down the venous return. The blood dammed up in the limbs is virtually removed from the circulation, and has the same effect as a venesection for the short period the tourniquets are left in position.

B. Acute Left Ventricular Failure

Here the patient is often admitted to hospital as an emergency in the middle of the night; he is more likely to be critically ill than the patient with congestive heart failure, and hours or even minutes count in his treatment. There is usually a history of breathlessness or angina of effort for months or years. In such a patient, symptoms of acute left ventricular failure may develop with dramatic suddenness. Often he starts out of sleep in the small hours of the morning, gasping for breath, blue or ashen grey, wheezing, and bringing up frothy sputum tinged pink. These are the symptoms of *acute pulmonary oedema*, resulting from left ventricular failure (see above). When wheezing is noticeable, such an attack is often called *cardiac asthma*.

Acute left ventricular failure occurs in hypertensive heart disease (p. 67), coronary artery disease (p. 71) and mitral and aortic valvular disease, whether rheumatic (p. 63) or syphilitic (p. 80). Attacks may be precipitated by anything which further weakens the damaged heart—infection, anaemia, or coronary occlusion. Sometimes it seems that pulmonary oedema occurs when the orthopnoeic patient slips down in the bed while he is asleep.

Treatment

Effective Treatment is a Matter of Urgency; everything should be got ready as soon as you hear that the patient is on his way.

(1) Have ready the *cardiac bed*, if one is available; otherwise the patient must be nursed propped up with a back rest and many pillows.

(2) *Oxygen* is given by the most readily available effective method (mask, bilateral nasal catheter or tent).

(3) *Morphia* ($\frac{1}{4}$ – $\frac{1}{2}$ gr.) is given to relieve breathlessness, distress and the anguish of mind these patients often suffer. (Contrast the treatment of chronic congestive heart failure, where morphia is given cautiously, if at all.)

(4) *Aminophylline* (0.25–0.5 Gr.) given by slow intravenous injection often has a dramatic effect in relieving wheezing and breathlessness.

(5) Venesection or limb tourniquets may be recommended in certain cases.

Treatment is then continued with oral aminophylline, digitalis and diuretics. Treatment of the underlying disease (e.g. hypertension) will often be necessary too.

Long-term Treatment of Heart Failure

The heart failure patient may spend two or three weeks in hospital, or much longer. Complete bed rest is necessary at first. During this time he should be encouraged to bend and stretch his legs to prevent venous thrombosis. As breathlessness subsides, oedema vanishes and the pulse rate returns toward normal he may be allowed out of bed for increasing periods.

After his discharge from hospital he usually rests at home for a time, or goes away to a convalescent home. He should get up late, go to bed early and rest in the middle of the day for a considerable period; he should also avoid any exertion which makes him severely breathless. Apart from this he should be encouraged to live as normal a life as possible. He may be able to return to work if his job is not too strenuous, or if he can change it for a lighter one. Digitalis treatment will often be continued indefinitely; a salt-low diet and diuretics will also be necessary in many cases. Obese patients should reduce their weight. Nurses can often help their patients by explaining the importance of continuing with the treatment recommended after discharge.

Prognosis.—This depends on the patient's heart, his circumstances and himself. The *heart* is usually permanently damaged to a greater or less extent, and the patient seldom recovers perfect health. The more severe the damage, the greater the danger of relapse. The patient will live longer if he can take things at his own pace and avoid all severe exertion. This depends partly on his circumstances—things are easier for a business executive with a bedroom on the ground floor than for a labourer in a third floor flat. But the prognosis in

heart disease also depends on the patient's temperament; the placid and philosophical man accepts restrictions while the restless and irritable person fights against them and in so doing succumbs.

Special Investigations in Heart Disease

(1) *X-ray of the Chest*.—This demonstrates enlargement of the heart and other abnormalities of the shape of the heart and great vessels.

(2) *Electro-cardiography*.—This demonstrates the electrical activity of the heart. The tracings are abnormal in many forms of heart disease. The electro-cardiogram (E.C.G.) is particularly valuable in the diagnosis of coronary occlusion (p. 75). This is because damage to the muscular wall of the heart, such as occurs in coronary occlusion, is apt to interfere with the normal spread of electrical impulses.

The other two investigations are much less frequently performed, and are carried out only when the results are likely to influence major treatment, e.g. in the selection of cases for cardiac surgery. They can only be carried out by those trained in the special techniques involved; they may involve discomfort or even risk for the patient.

(3) *Cardiac Catheterisation*.—A cardiac catheter is a very narrow tube which can be introduced into an arm vein. Under X-ray control the end is pushed up into the larger veins and finally into the right side of the heart; the pressure in the various parts of the right heart can be measured and samples of blood withdrawn for analysis (e.g. of oxygen content). Cardiac catheterisation is of value in the assessment of cases of mitral stenosis when surgery is being considered, and also in certain cases of congenital heart disease.

(4) *Angiography*. A substance which shows up on X-ray is injected intravenously and a series of X-rays of the heart rapidly taken. Abnormalities of the heart and great vessels can often be demonstrated, especially when there is a communication between right and left heart (e.g. patent interventricular septum, p. 83). Angiography is often useful in the diagnosis of the different varieties of congenital heart disease, especially when operation is under consideration.

(2) Rheumatic Fever, Chorea and Rheumatic Heart Disease

Although rheumatic fever is much less common than it was, it is still one of the most important of the diseases which attack the heart. In this country, hypertension and coronary artery disease are now much commoner, but they affect chiefly the elderly; rheumatic fever is a disease of youth, and the heart disease it causes all too frequently kills the patient in what ought to be the prime of life.

* * *

Acute rheumatism, or rheumatic fever, is a generalised infection of which heart disease is the most important but not the only result. The infection also attacks the joints, causing arthritis; chorea (St. Vitus' Dance) is another manifestation of acute rheumatism. As the effects of acute rheumatism may last a lifetime it is rarely possible to follow a patient throughout the various stages of his disease. It is like watching a play of which most of the early scenes are missing while the last act is repeated over and over again.

The nurse in the medical wards is likely to have in her care at one time or another cases of rheumatic heart failure, patients with recurrences of the rheumatic infection, those with old-standing heart disease being rested before an operation or confinement and—perhaps least commonly—cases of acute rheumatic fever. In the children's wards are to be found cases of acute and sub-acute rheumatism and chorea. These are some of the scenes which have now to be joined into one drama so that the plot can be followed through from beginning to end.

The Cause of Rheumatic Fever

An attack of rheumatic fever generally occurs one to three weeks after a *streptococcal throat infection* such as tonsillitis or scarlet fever. The original streptococcal infection is often trivial; why the body's reaction to it should be so abnormal is unknown. Only a very small proportion of streptococcal infections are followed by rheumatic fever.

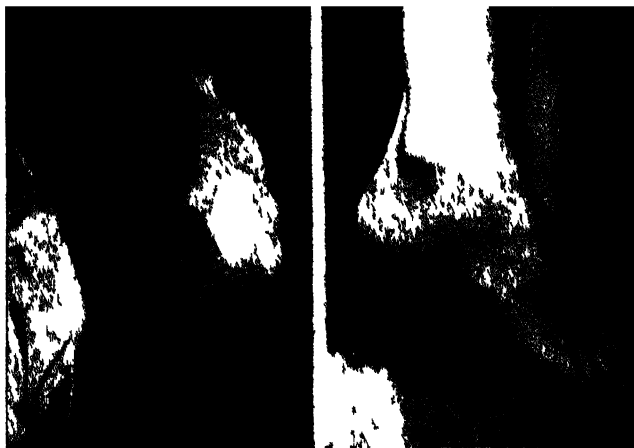


Fig. 17. Rheumatic Nodules on the elbow and ankle

Rheumatic fever is commoner among poor town dwellers than among country folk or the well-to-do. The Registrar General has shown that the death rate from rheumatic heart disease is twice as high in social class 5 as in the top social class, and towns are more heavily attacked than country districts. The reason is probably that bad housing and overcrowding facilitate the spread of streptococci, but poor food and other factors may be involved too. A person who has once had rheumatic fever is likely to have a recurrence after each attack of streptococcal sore throat.

Acute Rheumatic Fever

This disease chiefly attacks children and young adults; the peak incidence lies between 8 and 12 years of age. It is rare below the age of 5.

The main symptoms are *fever* and *joint pains*. In children the temperature is moderate to high, and in adults low fever is commoner. The *joint pains* vary from mild twinges to pain which is so severe that the patient cannot bear the limb to be touched. *Nose bleeding* is common, and various *rashes* may be seen.

On examination, the patient is usually pale and feverish, and the pulse is rapid and soft. In severe cases one or more joints are obviously inflamed—hot, reddened, swollen and acutely tender. The affected joints are usually those of medium size—wrists and ankles, elbows and knees; the spine, hips and fingers are usually spared. The signs of arthritis often flit from one joint to another over a period of days or weeks. The joints in rheumatic fever *always recover completely* in the end. In mild cases there are no signs of arthritis.

The Heart in Rheumatic Fever.—The patient seldom complains of heart symptoms during an acute attack, unless the uncommon and serious *pericarditis* develops. However, there may be signs that the heart has been affected—the rapid pulse may be out of all proportion to the degree of fever, and the raised pulse rate sometimes persists after the temperature has subsided. Abnormal signs may also be found with the stethoscope or electrocardiograph.

The Skin in Rheumatic Fever.—Rashes are commonest among children. The typical rash consists of pink blotches and spots which spread from the centre, leaving a clear area in the middle (*erythema marginatum*). *Rheumatic nodules* are also fairly common in severe cases in children. These nodules are small firm insensitive lumps, which can be felt under the skin, usually over bony prominences.

The Blood in Rheumatic Fever.—The *white cell count* is usually

raised, as in so many acute infections (see p. 156). The *blood sedimentation rate* is also raised—often markedly so. This test measures the rate at which red cells settle in a column of citrated blood. Normally the cells fall 3–10 mm. in a 200 mm. tube in one hour. A high B.S.R. is found in many diseases. In rheumatic fever a high B.S.R. means that the disease is still active; it falls as the patient recovers.

Some of the blood proteins are abnormal in rheumatic fever. An estimation of the *C reactive protein* in the blood is a more reliable test of activity than the B.S.R.

Course of Rheumatic Fever

All symptoms usually clear up in a matter of days or weeks. Signs of activity, such as a raised pulse rate, an abnormal electrocardiogram or a raised B.S.R., sometimes persist for much longer.

Treatment.—*Absolute rest* is all important whenever there is a danger of heart disease, and this means that there must be first class nursing. Bed rest continues as long as there are clear signs of active disease—say for a few weeks. Very long periods of bed rest are not usually recommended nowadays.

Drugs.—Large doses of *salicylates* have a dramatic effect in rheumatic fever; fever and arthritis clear up within a few days. The dose of salicylates is the largest the patient can tolerate—e.g. calcium aspirin gr. 20 four-hourly for an adult. Salicylates in this dosage are apt to cause nausea, vomiting and ringing in the ears, restlessness, flushing, sweating and overbreathing. Report any such symptoms to the doctor; they usually mean that the dose should be reduced.

Steroids (cortisone, prednisone, triamcinalone and allied drugs) also relieve the symptoms of acute rheumatic fever. In most cases the results are no better than those of salicylates. It is not yet known whether steroids help to prevent heart damage.

A Note on Cortisone and Allied Steroids

These drugs are either hormones which are produced by the cortex of the adrenal gland, or synthetic substances with similar effects. They have the remarkable property of suppressing the body's reactions to infective and allergic agents. Thus, symptoms such as fever, pain, swelling of tissues, and rashes are often relieved, and the patient feels much better. The underlying disease process, however, usually continues unchecked, and symptoms re-appear as soon as the drug is stopped, unless the disease is one, like rheumatic fever, which only lasts a few weeks. *Steroids do not cure any disease.* In the following diseases they suppress inflammatory

and allergic symptoms and produce at least temporary remissions: rheumatic fever, rheumatoid arthritis (p. 206), ulcerative colitis (p. 180), status asthmaticus (p. 136) and many skin diseases and drug reactions. In other diseases (nephrotic syndrome, p. 197, hæmolytic anæmia, p. 150, acute leukæmia, p. 158) their mode of action is more obscure. Finally, cortisone is given to replace the missing hormones in endocrine disorders such as Addison's disease (p. 234) and Simmonds' disease (p. 230).

Long-continued steroid treatment may have the following undesirable effects: obesity, acne, growth of facial hair (in women), œdema, hypertension, glycosuria, peptic ulceration, osteoporosis (softening of the bones) or mental disturbances. Also, it lowers the body's resistance to infection (e.g. pulmonary tuberculosis) and may mask completely the evidence of such infection until it is quite advanced. Cortisone and allied drugs also suppress the production of the natural hormones of the adrenal cortex. This means that if the drug is suddenly stopped the patient has no natural adrenal cortical hormones to fall back on and symptoms of acute shock may develop. *This is particularly likely to happen if the patient has an operation.* It is extremely important to know if surgical patients have had steroids within a year of admission to hospital; increased doses before and after the operation will then be necessary.

Because of the many undesirable side-effects of steroid therapy, these drugs are never given if simpler treatment is likely to be as effective. When steroids are given, the dose is kept as low as possible and the drugs stopped *gradually*, as soon as possible. When given during the course of a known infection, such as tuberculosis (p. 118) intensive chemotherapy to combat the infection must be given at the same time.

Chorea

It may seem strange that an illness in which neither fever nor arthritis occur, in which the symptoms are muscular weakness, involuntary movements and emotional upsets should be caused by the same infection as rheumatic fever. Yet such is the case. Over a quarter of choreic patients have already had acute rheumatism, nearly half develop it later. Most important of all, the heart is just as frequently affected and suffers in just the same way as in acute rheumatism.

Chorea chiefly attacks children between the ages of 10 and 15. The child seems to get clumsier and clumsier, weaker and more nervous. She is often scolded for "fidgeting" or for upsetting things and breaking china; she is irritable and nervous and apt to fly into tempers and bursts readily into tears. Then she starts twitching and grimacing, and soon the abnormal movements are generalised. The hands and face are usually affected most. The child often cannot feed herself and even swallowing may be difficult. The movements stop when she goes to sleep. An attack of chorea generally runs its course in from six weeks to six months, but second and third attacks are common.

Treatment.—The child is put to bed on a régime of absolute rest for a week, or longer if the heart is affected. If the movements are violent she should have a cot with padded sides. Sometimes children have to be fed through a nasal tube when swallowing becomes a difficulty.

Sedatives such as phenobarbitone or chloral help to keep these restless children quiet. Large doses of salicylates are given as in rheumatic fever.

In all cases of chorea, good feeding, a bright, cheerful atmosphere and long convalescence are essential.

Rheumatic Heart Disease

What is the outlook for the choreic child, or the youth with rheumatic fever? In either case the infection may be overcome, thanks to the patient's powers of resistance and to good nursing. The heart may escape infection altogether, or it may recover from a mild attack. But all too frequently, though the patient seems to make a complete recovery, the rheumatic infection is still there, insidiously infecting, scarring and finally crippling the heart.

What happens in such a case is this. In the acute stage signs of inflammation can be found in all parts of the heart-muscle, lining membrane (endocardium) and coverings (pericardium). In the later stages it is the lining of the heart which is most obviously and severely damaged. Special flaps of lining guard the openings between one chamber of the heart and the next. These flaps form *valves*, which prevent the blood from flowing backwards. The rheumatic infection is particularly liable to attack these valves, which become scarred, puckered and hardened. This deformity of the valves gravely interferes with the pumping action of the heart; the heart muscle has to pump blood against the resistance of a narrowed opening (stenosis), or the scarred valve flap may fail to close properly, so that some of the blood already pumped through flows back after each contraction of the heart muscle (regurgitation or incompetence). This muscle, itself damaged by rheumatism, is labouring under a double disadvantage. At first it may overcome the difficulty, it becomes thicker and heavier and works harder with each beat. But this process cannot go on indefinitely; little by little the heart fails to discharge with each beat the blood flowing in from the veins. The blood begins to accumulate in the dilated heart, then to stagnate in the veins of the lungs and the body, and signs of heart failure begin to appear.

There are many varieties of rheumatic valvular disease. The valves on the left side of the heart—the side which pumps blood round the whole body—are much more often attacked than those on the right. The *mitral valve*, guarding the opening between left auricle and ventricle, is most often affected; mitral stenosis and incompetence are the commonest forms of rheumatic heart disease. The *aortic valve*, between left ventricle and aorta, is another frequent victim; either stenosis or incompetence may result. More than one valve is often injured in the same patient.

Symptoms of Rheumatic Heart Disease.—Once the attack of acute rheumatism or chorea is over the patient may seem to be perfectly well for months or years while the rheumatic infection is silently corrupting the heart. Rheumatic heart disease is often found in people who have never had rheumatic fever or chorea, or who may have had some half-forgotten illness with “growing pains” many years before. In either case the symptoms of heart failure may come on insidiously when the patient is in his twenties or thirties. At first he notices shortness of breath on running upstairs; then walking uphill becomes difficult and finally the slightest exertion causes breathlessness. Sometimes the symptoms develop rapidly, especially when they are precipitated by illness, overwork or pregnancy.

Heart Failure in Rheumatic Heart Disease. The symptoms, signs and treatment of heart failure are similar whatever the cause and have already been described (see below for the operative treatment of mitral stenosis). In *mitral stenosis* the patient is apt to have attacks of breathlessness and recurrent pulmonary infection. The lips are often cyanosed and auricular fibrillation is particularly common. Another common symptom in mitral stenosis is *blood spitting* (hæmoptysis). Small hæmoptyses may be due to congestion of the lungs; more serious attacks may mean that a piece of clot has blocked an artery in the lung (pulmonary embolism). The failing heart often contains blood clot, and pieces of this clot are apt to break off and to be swept away into the circulation. Any artery may become blocked in this way; for instance, an embolism in the brain may occur, causing a *stroke*; embolism of a limb artery causes *gangrene* (in such cases it may be possible to remove the embolus at operation and so save the limb).

In *aortic valvular disease* the overworked left ventricle fails early, so that breathlessness is an early symptom and is very severe, and pulmonary œdema may develop. The cyanosis and œdema so common in mitral stenosis only appear in the late stages of aortic

disease. *Aortic regurgitation* can often be recognised by the *collapsing pulse* (p. 25).

Surgery in Mitral Stenosis

Every patient with mitral stenosis and with no other valve defect is now a possible candidate for the operation of *mitral valvotomy* (splitting the mitral valve). The operation may transform the life of a breathless patient and prevent further deterioration. The best results are obtained when the patient has good general health, slight to moderate symptoms, and no auricular fibrillation. However, mitral valvotomy can be successfully performed in the presence of auricular fibrillation and even pulmonary oedema. A severe degree of mitral regurgitation makes the operation useless.

It is occasionally possible to relieve *aortic stenosis* surgically, but this is a very much more difficult operation with a high mortality.

Outlook in Rheumatic Heart Disease. The patient who has enough money to buy good food and clothes and to live in a decent house or ground floor flat may live a more or less normal life for years. Once congestive heart failure has developed, however, the outlook is much more serious. Relapses always occur, and the patient may drift in and out of hospital for years, dying perhaps in his thirties or early forties—unless he is fortunate enough to have a successful operation for mitral stenosis.

Another danger is that of *bacterial endocarditis*, to be described later. This infection, which may attack the damaged valves of a rheumatic heart, may follow tooth extraction, which releases bacteria into the bloodstream. Penicillin is therefore prescribed before and after tooth extraction for those with rheumatic heart disease.

To sum up: acute rheumatism attacks principally young, poor town dwellers. The early attacks, whether of rheumatic fever or chorea, are apt to follow streptococcal infections. Although complete recovery may follow such attacks the heart is liable to permanent and incurable damage, resulting in heart failure, with disablement of varying degrees, often leading to death in youth or early middle age.

The Prevention of Rheumatic Fever

Prompt, intensive treatment of all streptococcal infections helps to prevent rheumatic fever. As those who have had rheumatic fever are so likely to have a relapse after each streptococcal infection many doctors seek to prevent such infections by prescribing oral penicillin

V or other preparation throughout the winter months—or even throughout the year.

Case Histories

(1) *Mrs. W., aged 41.*—Admitted to hospital having had severe breathlessness for a month. She had coughed up a cupful of blood a few weeks before. She had had rheumatic fever twice, aged 14 and 16. During her two pregnancies, when aged 25 and 27, she had had symptoms of heart failure and had twice been admitted to hospital since then with heart failure. *On admission* she showed signs of advanced congestive failure—too breathless to lie down, a rapid, regular pulse and signs of gross aortic and mitral disease. She improved with routine treatment and was discharged after five weeks in hospital. The outlook for this patient is very bad; she is unlikely to survive another attack of severe heart failure. Operation is impossible because of the aortic disease.

(2) *Mrs. A. aged 39,* had chorea as a child. She began to be breathless at the age of 30, and this breathlessness increased until she could not do the cooking and housework for herself and her husband (they lived in a caravan). She had marked swelling of the legs and orthopnoea. She was found to have mitral stenosis. Mitral valvotomy was advised and after much hesitation she agreed to the operation. Her breathlessness was much less after the operation, but she needed a great deal of encouragement to live a more active life. She can now however do the work of the caravan normally.

(3) *Mr. S., aged 42,* was found to have mitral stenosis and auricular fibrillation four years ago when he was suffering from a cough. He had never had rheumatic fever but remembers that his left knee and ankle became red and swollen for a week or two seventeen years ago. His fibrillation is well controlled with digitalis and he works fairly regularly as a carpenter. Recently he had a bad relapse after leaving off his digitalis for several weeks while he had a cold. At that time his pulse rate was 120 with a heart rate of 160. Six weeks' rest and digitalisation were needed before he was fit to go back to work. This man should be able to carry on quite well as long as he looks after himself, as he is not too badly off and is well cared for by a devoted wife.

(3) Hypertension

Hypertension, or high blood pressure, has been included in this section on heart disease because this disorder of circulation generally tells most severely on the heart. But hypertension, like acute rheumatism, leaves its mark on many organs. Rheumatism attacks heart, joints and brain; in hypertension, heart, brain and kidneys are the organs which suffer most. In both diseases heart disease is the most frequent cause of death.

In almost every other way hypertension is a contrast to rheumatic fever. Acute rheumatism attacks the young and the poor; hyper-

tension finds its victims among the elderly, the well fed and the worried. Rheumatic fever is on the decline—the rheumatic death rate fifty years ago was five times as high as it is to-day—whereas hypertension is on the increase. As the standard of living improves the conditions favouring acute rheumatism tend to disappear and, among an ageing, well-fed population, hypertension flourishes in its place.

The Blood Pressure—Normal and Abnormal

In hypertension the blood in the arteries is under an abnormally high pressure. The blood pressure, measured with the sphygmomanometer, is generally somewhere around 130/80 in a healthy young person—that is to say, the pressure is equal to that of 130 m.m. of mercury while the heart is contracting (the *systolic* blood pressure) and 80 m.m. while it relaxes (the *diastolic* pressure). The systolic pressure is very variable, even in healthy people; like the pulse and respiration rates it is raised by exercise and emotion. The blood pressure rises from about 100/60 in childhood to 150/90 or more in old age. Values above 170/90 are generally considered abnormal. Hypertensive patients commonly have blood pressures of 200/100 or more.

Causes of Hypertension.—The blood pressure is raised in many forms of kidney disease—e.g. nephritis (p. 190); pyelonephritis (p. 189); in toxæmia of pregnancy; in coarctation of the aorta (p. 83); and in certain endocrine disorders—e.g. Cushing's syndrome (p. 232), phæochromocytoma (p. 235). *In the vast majority of cases of hypertension the cause is unknown*; such cases are known as *essential* or *idiopathic hypertension*. But it is important to remember that hypertension *may* have a cause which can be discovered and treated (as by removal of a phaeochromocytoma, that is, a chromaffin tumour of the suprarenal gland).

The Hypertensive Patient

Hypertensive patients who are admitted to hospital are often suffering from the late effects of the disease—heart failure, stroke or kidney failure. Others are admitted for treatment with blood-pressure lowering drugs, or for investigation, in an attempt to discover one of the causes of hypertension mentioned above.

The hypertensive patient is often a stout, thick-set, middle-aged or elderly man who both overworks and overeats; he may also be a heavy smoker or drinker. Often other members of his family are hypertensive, and one of his parents may have died of a stroke.

Malignant Hypertension

This is a very severe form of hypertension, often affecting comparatively young people, in which there is a rapid and severe rise in blood pressure (e.g. to 250/150). Symptoms are severe, including defects of vision, for the retina is always affected in malignant hypertension and papillœdema is present. Often there are signs of kidney failure (see Chapter 13) and the patient may die in uræmia. In the absence of treatment the patient dies in a year or two. Hypotensive drugs may be life saving. The final prognosis is still serious.

Case History—Miss L., aged 44, had had severe headaches for 18 months, with giddiness, loss of power in the hands, black spots in front of the eyes and attacks of vomiting for two months. Her blood pressure was 250/180, her heart was enlarged, she had papillœdema, albumin in the urine, and blood tests gave evidence of advanced kidney disease. Venesection and sedatives gave temporary relief. Two months later she developed pulmonary œdema and died a fortnight later. (This was in the days before hypotensive drugs.)

Mr. P., aged 50, complained of headache and weakness of the legs; his blood pressure was found to be 210/140. He refused to stop work. A few months later he had a sub-conjunctival hæmorrhage; B.P. 250/140. Papillœdema was now found to be present. He was admitted to hospital; B.P. 300/205. He was treated with meclamine and reserpine; B.P. 180/100. He takes his own blood pressure (with the help of his wife) and adjusts the dose according to his findings. Health, good; papillœdema cleared up; patient is at work.

(4) Coronary Artery Disease and Angina Pectoris

The coronary arteries are those which supply the heart itself with blood. The heart cannot function without a proper blood supply; so disease of these arteries may have the most disastrous effects—from invalidism to sudden death.

Coronary artery disease is much commoner than it was a decade or two ago. In 1950 it caused over 91,970 deaths—about four times as many as in 1930. It tends to smite the elderly and the well fed, as does hypertension; and there are more elderly, well fed people in this and other prosperous countries than ever before. It is commoner among men than women, among rich than poor and among sedentary than active people. It is also commoner among smokers than non-smokers, and, like other forms of arterial degeneration (see p. 88) among diabetics than non-diabetics. Although the causation of coronary artery disease remains obscure it seems that a *diet rich in*

animal fat (butter, cream and lard), *lack of exercise*, and *heavy smoking* are at least partly responsible for the great increase in coronary artery disease among "civilised" people.

Angina Pectoris

The term means *narrowing of the chest* (angina means narrowing, not agony, as is often thought). The symptom most typical of angina pectoris is an attack of pain in which the chest feels as if it were *gripped in a vice*.

The anginal patient is usually a middle aged or elderly man, often one who is overweight and a heavy smoker. He describes how he is smitten by a pain in the chest quite suddenly as he hurries up hill or during some such exertion. The same amount of effort always brings on an attack—for instance, the pain may always come on as he passes the pillar-box half-way up the hill on the way to the station, or it may occur on the second, though not on the first flight of stairs. Cold, fatigue, over-eating and emotion all help to bring on an attack.

The pain which smites so suddenly on such occasions is most often felt in the centre of his chest. The whole chest may feel as if it were being crushed in a vice, and the pain, which may be slight or agonising, may spread up to the jaws or down the left arm. The victim is stopped in his tracks: motionless, he waits for the pain to leave him. As a rule the attack is over in a few moments. If seen during an attack the patient is found to be pale and sweating, but his pulse is little affected in rate and volume. The pain gone, he feels exhausted for a short time, but he recovers quickly, and is then free of pain until exercise brings on another attack.

The patient's story in angina is so characteristic that it usually settles the diagnosis. Clinical examination reveals little that is abnormal: the blood pressure is high in about half these patients. The electrocardiogram is often normal, though it may be abnormal, especially after exercise.

Cause of the Symptoms

The pain in angina pectoris is caused by an *inadequate blood supply to the heart muscle*. Here is an experiment which will help you to understand anginal pain. Put a tourniquet round your arm above the elbow and tighten it till the pulse can no longer be felt at the wrist. The limb feels uncomfortable but not painful. Now clench and unclench the fist a few times. Immediately a cramp-like pain is felt which prevents further exercise until the tourniquet is released.

In angina pectoris the blood supply to the heart muscle is adequate when the patient is at rest and the heart is beating quietly. But during exercise the heart beats more violently, the heart muscle has too little oxygen for its increased needs, and an attack of pain occurs.

The commonest cause of angina pectoris is *degenerative disease of the coronary arteries*. (This form of arterial disease is called *atheroma*). The arteries are partially blocked owing to degeneration of their walls. Anginal pain may also occur in *diseases of the heart and aorta*, which result in too little blood entering the coronary arteries (e.g. aortic stenosis or incompetence). Finally, *severe anaemia* may cause angina, as the heart (as well as other organs) receives too little oxygen even though the coronary arteries are normal.

Treatment

Treatment of the Anginal Attack.—The patient himself instinctively adopts the most important part of the treatment—he stops moving. After the pain has passed off he should, if possible, rest in a chair or sofa for about half an hour.

An anginal attack may be cut short by one of the *nitrites*, and the patient must never go about without a supply of one of these drugs. The nitrites dilate blood vessels all over the body; the blood pressure falls, giving the heart less work to do, and the coronary arteries dilate, improving the blood supply to the heart. *Amylnitrite* is a liquid which is put up in capsules which have to be broken and the vapour inhaled. The effect is almost immediate and is over in a few minutes. Amylnitrite is apt to cause uncomfortable flushing of the face, and throbbing and palpitation. *Glyceryl trinitrate* tablets (gr. 1/130) take effect in about two minutes and the effect lasts for 20 minutes. The patient should put a tablet under his tongue as soon as he feels an attack coming; the tablet is allowed to dissolve, not swallowed whole.

Prevention of Attacks.—As in all types of heart disease rest is the foundation stone of the treatment of angina. In severe cases—those who have attacks when walking on the level, or at rest—a period of rest in bed is indicated. In all cases the patient must learn to live within the limits of his powers. A quiet, regular life, with a rest after the main meal and eight or ten hours in bed at night is advisable. The patient should cut down all strenuous activity likely to precipitate an attack; he should avoid extreme cold, large meals, and should give up smoking. *Obesity* puts a big strain on the damaged heart; fat patients should keep to a strict reducing diet.

The *long-acting nitrites* can be taken to prevent anginal attacks—e.g. erythrol tetranitrate (gr. $\frac{1}{4}$), penta erythrityl tetranitrate ("mycardol," "peritrate") (gr. $\frac{1}{8}$ – $\frac{1}{4}$). The drugs are given in tablet form three or four times a day. They may be taken regularly, or only when the patient expects to be more active than usual.

The Outlook in Angina.—Patients with angina pectoris may suffer from a coronary occlusion, congestive heart failure or they may die suddenly. Among a very large series of anginal patients followed up at the Mayo clinic, U.S.A., it was found that 58 per cent. of cases survive for five years and 30 per cent. for ten years. By far the most important factor in assessing any one patient's prospects is the extent to which he is able and willing to adapt his life to his heart condition.

Coronary Occlusion

The same coronary arteries whose degeneration leads to angina pectoris are always in danger of becoming altogether blocked—a state of affairs known as *coronary occlusion*. About half the victims of the latter have previously suffered from angina, and all resemble the anginal group in age, sex and class. But whereas angina is often treated in Out Patients, patients with coronary occlusion need at least three weeks' absolute rest in bed, so that this condition is much more familiar to the nursing staff of a hospital ward than is angina pectoris. Indeed, few large male medical wards are ever without a case.

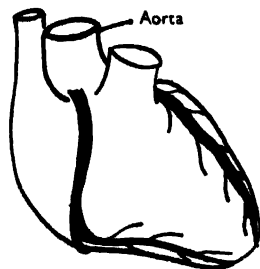


Fig 18. Diagram of the Heart and Coronary Arteries. The descending branch of the left coronary artery (shown on the right of the diagram) is most frequently blocked, cutting off the blood supply to part of the wall of the left ventricle.

A case of coronary occlusion is often admitted to hospital as an emergency, for the symptoms of this disease strike as suddenly as the pain of angina. The most prominent symptom is pain of the anginal type. The pain, however, generally comes on when the patient is at rest, and, instead of passing off in a few minutes, it may last for hours or even days. Rest does not relieve it—in fact, the patient often feels impelled to move restlessly about—nor do drugs of the nitrite group. In addition to the physical suffering the patient often endures great mental anguish and dread, with an acute feeling of approaching death. He will be found to be breathless, often blue, with a rapid, feeble, sometimes irregular pulse. In

severe cases he may be a greyish colour, cold, sweaty, anxious and restless, with an almost imperceptible pulse and a very low blood pressure. These symptoms of shock are absent in mild cases, and here the pain may be quite mild. Or pain may be absent, the patient having symptoms of heart failure of sudden onset.

These symptoms are all due to the sudden closure of a large branch of one of the coronary arteries, which deprives an extensive section of the heart muscle of blood. The blockage may be caused by clot formation or by a detached piece of degenerated arterial lining. The cutting off of the blood supply of part of the heart gives rise to pain, far more persistent than that of angina, though of the same nature. The breathlessness and circulatory collapse are due to the effects on the segment of heart muscle, which is at once put out of action and eventually dies.

Further Course of Coronary Occlusion

The first hours and days after a coronary occlusion are the most dangerous, and many deaths occur in these early stages. The damage to the heart muscle may precipitate *ventricular fibrillation*, which is rapidly fatal because it virtually stops the ventricles from pumping blood into the arteries; or there may be a second coronary occlusion; or congestive heart failure may develop; or the damaged heart may rupture, causing instant death.

If he survives these hazards the patient often makes a gradual recovery. A few days after the attack the temperature rises to 99° or 100°, and examination of the blood reveals an excess of white cells and a raised sedimentation rate. (The absorption into the blood stream of the breakdown products of dead heart muscle cause these disturbances.) The blood pressure begins to rise after a week or two, though it seldom reaches the pre-occlusion level.

If series of electro-cardiograms are taken characteristic changes will be found which not only clinch the diagnosis but also indicate exactly which part of the heart has been damaged.

Complications of Coronary Occlusion

Venous thrombosis is common in patients who have recently had a coronary occlusion, and may result in *pulmonary embolism*. Or a clot may form in the chamber of the heart itself, from which emboli may be carried to any artery in the body—e.g. in the brain or limbs.

Treatment of Coronary Occlusion.—It is clear that a man who has had a coronary occlusion, in whom there is a considerable risk of sudden death, needs the most skilful nursing. The patient must be

kept absolutely at rest until the damaged heart has had time to heal. He is propped up in bed, and disturbed as little as possible. Any slight strain or unnecessary exertion may be too much for the enfeebled heart muscle and may be the cause of sudden death. Thus, patients have died while straining at stool. For the first 48 hours after a coronary occlusion the bowels need not be moved; after that a small enema may be given with the utmost care every two or three days if the patient is constipated.

Morphia (gr. $\frac{1}{4}$ – $\frac{1}{2}$) is given to relieve pain and distress; *pethidine* (100 mgm.) may be substituted if morphia makes the patient sick. If the patient is blue he requires *oxygen*. *Heart failure* should be treated as already described.

Treatment of Shock.—If the blood pressure is extremely low (i.e. below 90/70) and does not respond to rest, warmth, morphia and oxygen, blood pressure raising drugs are sometimes given. *Nor-adrenaline* may be given slowly in a carefully regulated intravenous drip, or the less drastic mephentermine sulphate (another blood vessel constrictor) may be given by intramuscular or intravenous injection. These drugs can be life saving in severe shock, but they are dangerous in that they may precipitate serious cardiac irregularities such as ventricular fibrillation. The blood pressure should be taken every 15–30 minutes during this treatment.

Anticoagulant Treatment.—As we have seen, patients who have had a coronary occlusion, may have further coronary thrombosis, venous thrombosis, pulmonary embolism or other arterial emboli. To prevent these complications *anticoagulants* are given—drugs which reduce the clotting power of the blood. These drugs are usually prescribed in all but the mildest cases. At the start of treatment an injection of *heparin* (20,000 units) is given. This takes effect almost immediately. At the same time an oral dose of ethyl biscoumacetate (“tromexan”) or phenindione (“dindevan”) is given. These drugs take effect more slowly and their action is more prolonged. Frequent blood tests are necessary to control treatment with anticoagulants; the test is called the *prothrombin time*. The dosage is adjusted according to the results of the prothrombin time tests. If too much anticoagulant is given the *patient is liable to bleed*. Nurses should watch their patients carefully for bruising, hæmaturia, rectal bleeding or nose-bleeding, and must at once report any such bleeding to the doctor.

Anticoagulant treatment is *never* given unless regular prothrombin time estimations are possible.

Anticoagulants are given, if at all, throughout the patient's stay

in hospital and often for long afterwards. Some physicians believe that they should be continued indefinitely. Blood is taken for prothrombin time estimation every 3-4 weeks as long as treatment continues. The patient may come up to Out-Patients for the test, or the family doctor may take the blood and send it to the laboratory.

Further Treatment.—The patient usually stays in hospital for 3 to 6 weeks. Once the acute stage is over he should be encouraged to bend and stretch his legs in bed to prevent venous thrombosis. After discharge from hospital he requires several weeks' convalescence or rest at home. Even then, he must reduce his activities and learn to live within the limits of his strength. If he is overweight a reducing diet is a necessity; obesity shortens life in coronary artery disease.

The outlook in coronary occlusion.—As with other forms of heart disease this is much better if the patient is able and willing to adapt his life to his reduced powers. Those who survive the first attack seldom recover their full vigour. Moreover, their coronaries are still diseased and further branches may become blocked at any time. Death is generally due to another attack of coronary occlusion. In some cases angina pectoris or congestive heart failure supervene. About half those who survive the first attack are still alive two to three years later. Exceptionally, a man may survive for many years with unimpaired vitality. The great eighteenth-century surgeon, John Hunter, suffered two attacks of coronary occlusion ten and twenty years before he died, and he was a man of amazing vigour. His death eventually occurred during a hospital Board Meeting, where the great man was infuriated by a fellow member of the Board.*

Case History.—Dr. E., aged 60, a busy general practitioner, overweight, heavy smoker, was smitten with pain in the chest while listening to the wireless one evening. Severe breathlessness and distress. Admitted to hospital; in great pain; slightly cyanosed; B.P. 150/110. Treated with morphia, oxygen, heparin, phenindione. Made good recovery in six weeks. Returned to part-time work within three months. Phenindione treatment continued, samples of blood being sent up to laboratory at regular intervals (weekly at first, then fortnightly).

(5) Cor Pulmonale

This is a heart disorder which is secondary to chronic lung disease—usually chronic bronchitis and emphysema, less often long standing pulmonary tuberculosis, bronchiectasis or pneumoconiosis. In some

*Hunter once said to a friend, "My life is at the mercy of any fool who chooses to plague and tease me." He was a hot tempered man and he rightly thought that a fit of rage would be the death of him. Anger—or any other violent emotion—is bad for these cases because it sends the blood pressure up and puts extra work on the heart.

hospitals cor pulmonale is the cause of about one third of all cases of congestive heart failure.

Symptoms.—The symptoms of chronic chest trouble often shade imperceptibly into those of congestive heart failure. The patient has usually been short of breath for years. *Cyanosis* is also attributable in the first place to the lung disease. Cyanosis of the lips and tongue is particularly pronounced. At this stage the pulse is of good volume and is usually regular; the hands and feet feel warm.

Later, the patient becomes more severely breathless and his feet swell. He is often drowsy and confused, and his fingers twitch; at length he may sink into coma. The pulse rate rises and the volume of the pulse is poor, but the rhythm usually remains regular. The hands and feet now feel cold to the touch.

These symptoms may develop insidiously, or they may be precipitated by an acute respiratory infection such as bronchopneumonia.

Explanation of the Symptoms.—In chronic lung disease much of the lung tissue may be destroyed, and the pulmonary vessels are obliterated or obstructed. This obstruction increases the work of the right ventricle, and, perhaps after many years, right ventricular failure occurs, causing the usual symptoms of congestive heart failure. The special features of cor pulmonale heart failure—early and severe cyanosis, drowsiness and muscle twitching—are the result of severe *oxygen lack* and *carbon dioxide excess*, for in the diseased lung the blood cannot be properly oxygenated, nor CO₂ discharged. CO₂ poisoning causes muscle twitching, stupor and finally coma.

Treatment.—*Antibiotics* are usually necessary for the respiratory infection which precipitated heart failure. *Antispasmodics* such as aminophylline—intravenously if necessary—are given if there is much wheezing. *Digitalis* and *diuretics* are given as in other cases of congestive failure. *Sedatives* are given only if absolutely necessary, as they are apt to depress respiration still further. *Paraldehyde* is often the safest. Morphia, a dangerous respiratory depressor, is never given.

Oxygen.—These patients are severely anoxic (short of oxygen) and one might suppose that oxygen would be the first necessity of treatment. Curiously enough, however, too much oxygen may depress the respiration still further, and precipitate CO₂ poisoning. The reason for this is as follows. Normally, any increase of the CO₂ in the blood stimulates the centre in the brain which controls respiration, and as a result the depth and rate of respiration are increased. In these patients, however, there is such a large excess of CO₂ that the respiratory centre is poisoned, and will no longer respond if the

may break off and, carried away in the circulation to some distant part of the body, may there block an artery. If the artery is an important one—say, a large one in the brain—the embolism may be fatal.

A patient with bacterial endocarditis often shows signs of both septicaemia and embolism. His temperature is high and irregular, he is pale, with a muddy complexion, and he wastes rapidly. The pulse is rapid and feeble, and the breathing quickened. All these are symptoms of septicaemia. Clubbing of fingers may also occur. Sometimes the onset is very insidious—especially in old people—and the patient may be admitted to hospital as a case of anaemia, loss of weight or low fever of unknown cause.

The signs of *embolism* are many and varied. A large artery in the brain or a limb may be blocked, resulting in paralysis or gangrene. Blood-stained urine or a sudden pain in the left loin may be caused by embolism of a large artery to kidney or spleen. Small arteries as well as large may be blocked. Crops of tiny purple spots may be scattered over the skin (purpura) when the small skin arteries are affected. If the small branches of the kidney artery are blocked the urine will contain blood cells, though a microscope may be necessary to find them. This is a most important sign of the disease in doubtful cases.

The diagnosis is often difficult in the early stages; it is clinched if organisms can be cultured from the blood.

Treatment.—These patients are often desperately ill as they are suffering from heart disease as well as a severe infection. So they need the most expert nursing on both accounts.

Bacterial endocarditis was always fatal before the discovery of penicillin. In the majority of cases enormous doses of penicillin will control the infection—up to five million units a day or even more for six weeks. A combination of penicillin and streptomycin is necessary in some types of infection. Other antibiotics may be given if the organism is penicillin resistant. Relapses may, however, occur.

It must be remembered that, even when the infection is cured, these patients still have damaged hearts, and hence will need the usual treatment of heart disease, such as has been described so often in the past few chapters.

Case History.—J.G., aged 29, a draughtsman, was sent up to the Chest Clinic because he had lost weight and felt tired and depressed. His temperature was 99°; he looked ill and anaemic. He had signs suggestive of congenital heart disease; his lungs were clear on X-ray. He was admitted to hospital. His temperature was irregular for a week. Blood culture grew *strep. haemolyticus*. He recovered after 6 weeks' treatment with penicillin (1 million units a day).

A note on Penicillin and other Antibiotics*

The discovery of penicillin by Sir Alexander Fleming, and its development by Sir Howard and Lady Florey during the Second World War, opened a new era in the treatment of many infections. Fleming noted that staphylococci did not grow near a mould—*penicillium notatum*—which appeared by chance on one of his culture plates at St. Mary's Hospital. This was because the mould produced *penicillin*, and this both killed bacteria and inhibited their growth. Years passed before the Floreys discovered how to produce a concentrated preparation which could be used in treatment. Penicillin has been in general use in this country since 1946.

Penicillin cures many infections and is seldom toxic. It may cure when all older remedies fail, as in bacterial endocarditis. Many diseases are more rapidly and safely cured by penicillin than by other drugs—e.g. syphilis, gonorrhœa, pneumonia, otitis media. Wound infections, eye infections and many other conditions may respond rapidly to penicillin.

Unfortunately, many organisms in time become resistant to its onslaughts, so that diseases caused by these particular strains can no longer be cured by penicillin. We have already seen (p. 42) that many ward infections these days are caused by penicillin-resistant organisms, most of the susceptible strains having been weeded out.

Penicillin is usually given by intramuscular injection. *Crystalline penicillin* has to be given every 6 to 12 hours, as it is rapidly excreted. *Procaine penicillin* is more slowly absorbed from the site of injection, and so

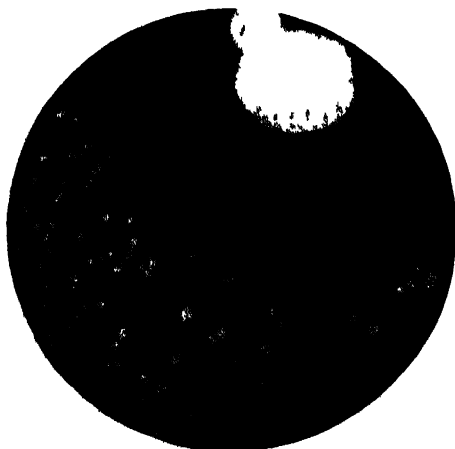


Fig. 22. *Penicillium notatum*.

Sir Alexander Fleming's original culture. The large white patch at the top is the mould *penicillium*; the smaller white blobs are colonies of staphylococci. Note that colonies are almost invisible near the patch of mould. For a long time all penicillin all over the world was manufactured from descendants of this very culture.

**Antibiotics*: anti, against; bios, life. The word means that a living organism (e.g. a fungus) antagonises another living organism (e.g. a disease bacterium). Antibiotics used in medicine are drugs derived from fungi. The term is often used (inaccurately), for any drug used to combat infection.

only one or two injections in the twenty-four hours are necessary. *Benzathine* is a very slowly acting penicillin; the effects of an injection may last a month. It is sometimes used in the prevention of rheumatic fever (p. 64). Unfortunately, local reactions are common and sometimes severe.

Oral penicillin is often satisfactory in the less severe infections; absorption is less reliable so larger doses have to be given.

Dosage. As a rule, at least 300,000 units of penicillin a day are given, often much more. In most acute infections a course only lasts a few days.

Toxic effects. These are rare. Patients who have been treated with penicillin in the past very occasionally collapse after an injection—*anaphylactic shock*. *Rashes* may occur, especially if penicillin is used locally in skin infections. Nurses who give many injections of penicillin may become sensitised and develop dermatitis of the hands and face. To prevent penicillin coming into contact with the skin one should use only a non-leaky syringe and express the bubble of air from the syringe while the needle is still in the ampoule. Wash immediately after giving the injection. Once dermatitis has developed it is difficult to treat and even the wearing of rubber gloves may not prevent relapses.

Other antibiotics. Streptomycin, chloramphenicol (chloromycetin), chlortetracycline (aureomycin), oxytetracycline (terramycin), tetracycline, erythromycin and novobiocin are prepared from other species of fungus. They are used in infections which do not respond to penicillin. Streptomycin is chiefly used in tuberculosis (p. 117). Chloramphenicol is of value in typhoid fever (p. 319) and is used in chest and urinary infections which do not respond to other drugs. The tetracyclines may be given in infections caused by penicillin-resistant organisms. They can all be given by mouth. Preparations for injection are also available for comatose or vomiting patients. These antibiotics clear the body so drastically of bacteria, those normally present as well as disease-producers, that fungi such as that of thrush may move in and flourish in their place; sometimes these fungal infections—of mouth, bowel or lung—are dangerous. A severe—often fatal—variety of enteritis caused by antibiotic-resistant staphylococci may also follow the use of these drugs, especially in post-operative cases. *Chloramphenicol* may cause aplastic anæmia (p. 149), but usually only when prolonged courses are given.

Drug resistance is a problem with these antibiotics, just as it is with penicillin.

(9) Pericarditis

This is an *inflammation of the pericardium*—the membrane covering the heart. The commonest variety of pericarditis is that which may occur during an attack of *acute rheumatism* (p. 59). Pericarditis may complicate *pneumonia*, *scarlet fever*, *tuberculosis* and other infections; it occurs in *septicæmia* and *pyæmia* and, occasionally, in patients dying of *uraemia* or *cancer*.

In acute pericarditis the patient usually complains of pain in the front of the chest; he is feverish, short of breath, and looks ill. The

pain is caused by the rubbing together of the two layers of inflamed pericardium. This stage is called *acute fibrinous pericarditis*; later on serous fluid may collect between the two layers of pericardium (sero-fibrinous pericarditis). The patient is uncomfortable and restless, blue and breathless. If there is a big effusion, the pulse is rapid and may almost disappear whenever the patient breathes in (*pulsus paradoxus*). In children, the distended pericardium may bulge out the front of the chest.

In septic conditions the pericardium may contain *pus* (purulent pericarditis). The patient is then very ill and breathless with high, swinging temperature.

Treatment.—The general treatment of fibrinous pericarditis is the same as that of rheumatic carditis (p. 60). If there is much pain over the heart, poultices or icebags may be applied. If much fluid collects the patient may be too breathless to lie flat, when a *reclining* position will be more comfortable. It is sometimes necessary to draw off the fluid. *Cortisone* may be effective in rheumatic pericarditis.

Purulent pericarditis calls for treatment with antibiotics; surgical drainage may be necessary.

Chronic constrictive pericarditis may follow tuberculous and other infections of the pericardium (rarely, if ever, rheumatic pericarditis). The heart is encased in a hard unyielding covering, and congestive heart failure results. Surgical treatment is often successful.

Prognosis. Most patients with pericarditis recover, though it is one of the most serious complications of rheumatic fever. The outlook in *purulent pericarditis* is grave.

(10) Arterial Disease

Arteries are the channels through which blood flows from the heart to the rest of the body. If an artery is narrowed or blocked, the blood flow to the tissues it supplies will be reduced or cut off. Most of the symptoms of arterial disease are those of *ischæmia* (lack of blood).

(a) Raynaud's Disease

The patient is usually a woman, who suffers from attacks of "dead fingers" in cold weather. The fingers go white or blue when cold, and feel numb and awkward; on warming they go bright red and tingle uncomfortably. Attacks often occur several times a day in cold weather; they tend to disappear during pregnancy. The feet may be affected as well as the hands. In most cases Raynaud's disease is simply a nuisance. Rarely, the circulation does not return to normal

between attacks and, in severe cases, gangrene of the finger tips may occur.

Blanching of the fingers in Raynaud's disease is due to *spasm of the local arteries*; the spasm is probably caused by overaction of the *sympathetic nerves*, which cause constriction of small arteries. The cause of the sympathetic overaction is unknown.

Treatment. Warm clothing should be worn—especially warm gloves and stockings—in cold weather. Vasodilator drugs such as tolazoline ("Priscol") often prevent attacks. In the rare, severe cases, when gangrene threatens, the ganglion supplying sympathetic nerves to the limb may be excised. This ganglion—the *stellate ganglion*—is in the neck. The operation is called *cervical sympathectomy*. It may successfully relieve arterial spasm in Raynaud's disease.

(b) Atheroma (Atherosclerosis)

This is a disease of the arterial lining or *intima*. It occurs mainly, but not exclusively, in the elderly. It is particularly common among diabetics; it is also common in hypertension. Atheroma may affect any artery in the body; it is the main cause of coronary artery disease (p. 71) and cerebral vascular disease (p. 282). Here we are concerned with atheroma of the peripheral or limb arteries. The *leg arteries* are much more often affected than those of the arms.

Symptoms include numbness, tingling and burning pains in the toes, cramps and weakness of the foot and leg. *Intermittent claudication* may also occur. Here attacks of cramp-like pain—usually in the calf—are brought on by exercise. As in angina pectoris, the pain starts suddenly after a certain amount of exercise—say, after walking half a mile. The patient stops dead in his tracks and waits for the pain to subside. As the disease progresses, the pain is brought on by less and less exercise.

On examination the foot feels cold to the touch; the skin often looks shiny and tightly stretched, and the tips of the toes may be discoloured. No pulsation can be felt in the *arteria dorsalis pedis*. (This artery can normally be felt between the extensor tendons to the first and second toes.) In severe cases *gangrene* develops in one or more toes, or at the site of some injury, such as a badly-cut corn.

Investigations

(1) The patient's *arms* are warmed by immersing them in hot water. The temperature of the skin of the affected foot is measured

with a special skin thermometer. If the skin temperature *rises*, this shows that the blood vessels are capable of expansion and are not completely blocked.

(2) Radio-opaque material is injected into the main artery to the

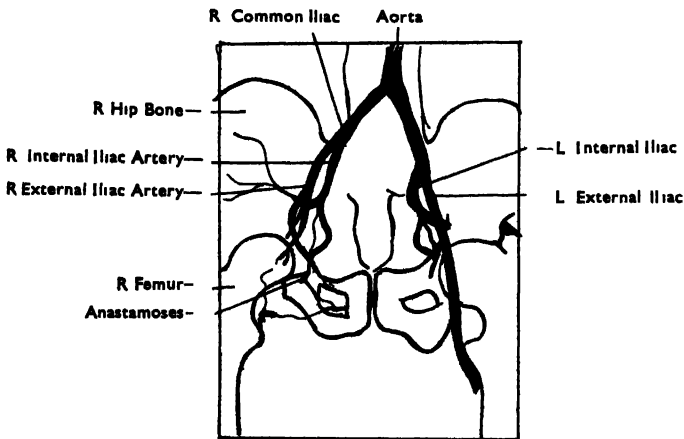


Fig 23 Arterial Occlusion.

Arteriogram showing occlusion of the R external iliac artery which tapers out shortly after the common iliac artery divides into internal and external branches. Note the anastomoses (joining up) of branches of the internal and external iliac arteries. These anastomoses supply tissues below the block with blood. The external iliac artery carries the main blood supply to the lower limb. The arteries on the left are normal.

limb and X-rays are then taken of the limb. The position and extent of any arterial block can be demonstrated in this way.

Explanation of Symptoms.—In atheroma little plaques of fatty substances develop in the arterial lining. The thickening of the intima may slow down or block the flow of blood along the artery, or a clot may start from one of the fatty plaques and eventually block the vessel. Unless there are enough subsidiary vessels (*collaterals*) to carry on the circulation the tissues supplied by the blocked artery die and become gangrenous.

Treatment

General.—*Smoking* should be forbidden (tobacco causes vaso-constriction). *Diabetes*, if present, requires careful control. Some doctors recommend low fat diets in atheroma. In the early stages the patient is advised to protect his feet and legs against injury, cold and heat (e.g. he should not sit with his feet close to the fire). If pain is severe and gangrene threatens the patient is kept in bed with the foot of the bed *low*; this improves the circulation. Heating the legs, as with hot water bottles, is forbidden; it increases the likelihood of tissue damage. But *heating the trunk* is often valuable; it improves the circulation in the legs by reflex action. Sometimes the circulation is improved by alternately raising the legs above and lowering them below heart level (Buerger's exercises).

Drugs.—Vasodilators such as tolazoline are rarely helpful, as the arteries are blocked rather than spastic. However, they may be tried.

Surgery.—For the same reason, sympathectomy is usually, though not always, ineffective. If the warming test described above is positive, sympathectomy is more likely to be successful in abolishing arterial spasm and improving the circulation. If a sudden blockage due to embolism occurs it is now customary to cut down on the artery and remove the embolus, thus restoring the circulation. If this is not done gangrene develops and amputation, especially in diabetics, is often necessary.

(c) Thromboangiitis Obliterans (Buerger's Disease)

This disease mainly affects men aged 20 to 40, whereas atheroma of the leg arteries rarely causes symptoms below the age of 50. The patient is often a heavy smoker. Jews are affected more often than those of other races.

The symptoms are similar to those described above. Intermittent claudication, however, is commoner, and may be present for years before gangrene occurs.

Treatment is similar to that already described under Atheroma.

N.B.—Arteriosclerosis. (Hardening of the arteries.) This term includes a variety of degenerative conditions in which the artery walls are thick and hard, including atheroma and the symptomless hardening of normal old age.

(II) Venous Thrombosis

Whenever the blood stagnates in the vessels it is apt to clot, especially if the veins themselves have been damaged by circulating toxins. Venous thrombosis may occur in illnesses such as pneumonia or typhoid, after operation or childbirth, or in any illness which



Fig 24 Venous Thrombosis.
Here there is thrombosis of the deep veins of the right calf, shown by distended superficial veins and slight swelling of the leg (infra-red photography) Dr Keith Ball's Case

keeps the patient long in bed. Nurses should be on the lookout for signs of venous thrombosis in such cases. Suspicious early signs are: an unexplained rise of temperature and pulse rate, restlessness or uneasiness, or pains and cramp in the calf. Such symptoms should at once be reported to the doctor. At this stage the leg may be slightly swollen and bluish, the skin glossy and the superficial veins distended (Fig. 24). Later, the whole leg may swell up like a tense,

white sausage (*white leg*). This means that the main femoral or iliac vein is thrombosed.

Treatment.—The patient is kept in bed and the limb protected with cotton-wool. Anti-clotting drugs such as phenindione may be given; they prevent extension of the clot. After a few days the patient is encouraged to bend and stretch his leg in bed; he gets up after ten to twenty days, according to the severity of the case, wearing an elastic stocking or supporting bandage. If there is much oedema, diuretics such as chlorothiazide may help.

Complications of Venous Thrombosis. After an attack of venous thrombosis ulcers of the leg are apt to develop (see chronic leg ulcer, p. 276). More dangerous, however, is *pulmonary embolism* (p. 128), when a piece of clot breaks off and is carried away in the blood stream and lodges in a lung artery.

Prevention of Venous Thrombosis. Blood is likely to clot whenever it stagnates, so, to prevent clotting in the leg veins, patients who have to spend a long time in bed should be encouraged to bend and stretch their legs and to turn over in bed from time to time, instead of lying like a log. If they are too weak to move themselves, the nurses should move them. Venous thrombosis is uncommon where patients get up as early as possible after operation, childbirth, etc.

Phlebitis. (Inflammation of the vein.) Pain and tenderness along the course of the affected vein occur; the overlying skin is reddened. *Treatment* is that of the thrombosis which always occurs in the inflamed vein.

Embolism. To conclude, here is a summary of the causes of embolism.

Embolism means the blocking of an artery by material which is carried by the circulation. Most often the material is a piece of blood clot, but *fat embolism* may occur after a fracture (the fat coming from the bone marrow) or *air embolism* if, for instance, a pneumothorax needle by accident enters a vein.

Pulmonary embolism (p. 128) is usually caused by a piece of clot originating in a leg vein, as described above. Embolism of other arteries (e.g. cerebral embolism, p. 286; embolism of a limb artery) is usually caused by clot arising in the heart, for the failing heart—in particular, the fibrillating auricles—is apt to contain blood clot. Pieces of diseased valve in bacterial endocarditis (p. 85) may also give rise to embolism.

CHAPTER TEN

DISEASES OF THE CHEST

(1) Pneumonia

THE word "pneumonia" includes several varieties of acute inflammation of the lung. The two commonest varieties are *lobar pneumonia*, in which a whole lobe or a large portion of one lung is uniformly inflamed, and *bronchopneumonia*, where the inflammation is more patchy and more often scattered through both lungs.

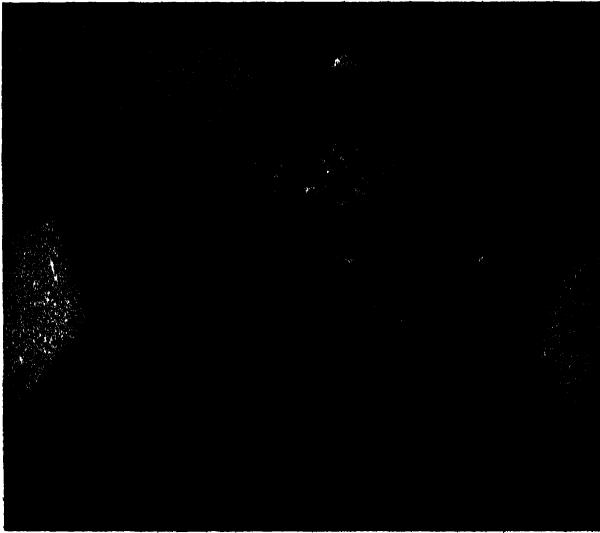


Fig. 25. Herpes of the lips (*Herpes Labialis*)
Also known as *Herpes Febrilis* when it occurs in a patient with fever
From A. C. Roxburgh's *Common Skin Diseases*. (H. K. Lewis)

Lobar Pneumonia

Lobar pneumonia may occur at any age, but it is most common between the ages of 15 and 40. It occurs principally in the winter, from November to March and, though it may smite the young and the robust, it attacks more frequently and more vehemently the enfeebled and the under-nourished. It is two or three times as common among men as among women.

Lobar pneumonia generally comes on suddenly with fever, shivering and a rigor. Vomiting is also a common early symptom, and, in children, there may be convulsions. Within a few hours the temperature has risen several degrees, and a short, dry, distressing cough quickly develops, with pain on one side of the chest. This pain is increased by coughing or by taking a deep breath. By the time the patient is admitted to hospital, usually on the first or second day of the disease, he presents a characteristic appearance. He lies on his back or on the painful side. His face is flushed, eyes bright, his skin is hot and dry. On his lips there may be a crop of little blisters and sores known as *herpes*, and his tongue is thickly coated with a white fur. His temperature is high— 103° or more—and his pulse rapid and, in severe cases, feeble or even irregular. In such cases the lips and cheeks will have a bluish tinge. His rapid, shallow breathing is often accompanied by the working of the nostrils, and expiration may end in a gasp or grunt. Every now and then he gives a painful cough, without expectorating any sputum at this early stage.

About the second or third day of the disease the patient usually begins to bring up a little scanty, sticky, brownish sputum which becomes more copious as his cough loosens.

Most pneumonia patients are sleepless with pain and fever and many are delirious. Some restlessness and distress are nearly always present.

Later on, if treatment is successful, the temperature falls rapidly to normal, the pain diminishes and the patient feels much better, though still weak. (See Fig. 4, p. 17.) In pre-sulphonamide days the temperature remained at a high level for a week or more, when it characteristically fell rapidly to normal—the *crisis* of the disease.

Complications.—*Heart failure* may occur in severe cases, the heart muscle being poisoned by bacterial toxins. Some degree of *pleurisy*—inflammation of the pleura—is almost invariable; sometimes this pleurisy may go on to pus formation—*empyema* (p. 98). Occasionally infection spreads via the blood stream to other parts of the body, causing meningitis, peritonitis, otitis media, etc.

Explanation of Symptoms.—The symptoms and signs in lobar pneumonia are the result of an acute bacterial infection causing inflammation of part of the lung and of its pleural covering. The fever and rapid pulse, the furred tongue and the herpes on the lips are due to the bacterial toxæmia (toxins in the blood). The rapid breathing and the harsh cough are caused by the pulmonary condi-

tion, for the affected part of the lung becomes solid, airless and inelastic (*consolidation*) as the air sacs fill up with inflammatory cells and exudates. These cells and exudates form the scanty *sputum*, which is brownish owing to the presence of altered blood. The *pain* in pneumonia is caused by friction between the two layers of inflamed pleura covering the affected part of the lung.

Special Tests in Pneumonia.—The responsible organism may be found in the sputum and is usually the *pneumococcus*. *Blood counts* show that, in response to the bacterial invasion, the white blood corpuscles, those defenders of the body against infection, have become very numerous—20,000–30,000 per cubic millimeter instead of the usual 10,000. *Blood cultures* may show that the pneumococci are actually present in the blood in severe cases. *X-rays* confirm the clinical signs of consolidation (solid lung).

Treatment.—*Good nursing is absolutely essential* and may be more important even than drug treatment. Absolute rest, physical and mental, is necessary. Neither nurse nor doctor should disturb the patient except when it is essential to do so. The ward should be airy but not too cold. A kaolin poultice to the affected side of the chest is often soothing, but it needs renewing every few hours if it is to be kept hot, and it may not be worth the disturbance to the patient.

The diet should consist largely of fluids and semi-solids at first—milk, eggs, custards, soup and so on. At least five pints of fluid daily must be insisted on if the patient is being given sulphonamide treatment. The diet can be increased as the appetite returns.

A sedative is generally necessary for the insomnia, for sleep must be secured at all costs in pneumonia. Pethidine (100 mgm.) relieves the pleural pain and is generally given at night for the first 48 hours or so. A hypnotic is often needed as well—paraldehyde, chloral or a barbiturate. Oxygen may be necessary if the patient is cyanosed or very breathless. If the pulse is irregular digitalis will be required.

Chemotherapy in Lobar Pneumonia (a) *Sulphonamides*. Most mild or moderately severe cases of pneumonia respond to sulphonamides in large doses (see below). As a rule the temperature reaches normal within two or three days, after which recovery is rapid, unless the patient is old or debilitated.

(b) *Penicillin*. This drug usually cures pneumonia more rapidly than do the sulphonamides, and it is used in most severe cases, at least at the beginning of treatment. In very severe cases frequent injections of crystalline penicillin are given. In less severe cases pro-

caine penicillin is given every twelve or twenty-four hours; sulphonamides are often administered at the same time.

Fortunately, most of the organisms causing lobar pneumonia are penicillin sensitive. If there is no response to penicillin and/or sulphonamides blood and sputum tests may identify the responsible organism and also the antibiotics to which it is sensitive. One of these is then prescribed.

Convalescence.—This is generally rapid, but a constant look-out must be maintained for complications during the first week or two. In elderly patients signs of heart failure may appear when they first get up.

A Note on the Sulphonamides

The first drug ever shown to be curative in pneumonia was *sulphapyridine* ("M & B 693"). Its action was first demonstrated at the Dudley Road Hospital, Birmingham, where, in 1938, Drs. Evans and Gaisford found that only eight out of a hundred cases of pneumonia treated with sulphapyridine died, compared with twenty-seven deaths among a hundred untreated cases.

Sulphapyridine was not the first sulphonamide to be discovered. This was *sulphanilamide*, which was active against streptococci and some other organisms, but useless in pneumonia. These drugs have largely been supplanted by the more potent sulphadiazine, sulphadimidine and sulphamerazine.

The discovery of the sulphonamides marked the beginning of a new epoch in medicine. Previously no drug had been discovered which would destroy disease-producing organisms in the body without harming the patient, with the exception of quinine in malaria, arsenic in syphilis and a very few others. The action of the sulphonamides is to prevent the growth of bacteria, so that the body's own defences can overcome the invasion. Sulphonamide treatment has proved successful in pneumonia, meningitis, urinary infections, erysipelas, tonsillitis, otitis media and many other infections and has by no means been superseded by antibiotics.

Administration and dosage. Sulphonamides are usually prescribed in tablet form (0.5 gramme). Emulsions can be prepared for children. For adults, one gramme of sulphadiazine four hourly, one gramme of sulphadimidine six hourly, or one gramme of sulphamerazine eight hourly, is usually required. A double dose is given at the onset of treatment.

The chosen drug must be given regularly *day and night*, to make sure that, throughout the twenty-four hours, enough is present in the blood stream to combat the infection. Generally a course lasts for five to seven days.

While the patient is under treatment he should take at least five pints of fluid daily, to guard against kidney damage (see below).

Toxic effects. The most important is kidney damage. The sulphonamides are excreted by the kidney and, if the patient does not drink enough, crystals of the drug may clog the kidney tubules and ureters, causing

hæmaturia and, in severe cases, suppression of urine. Report to the doctor immediately if any patient's urine becomes scanty or blood-stained while he is having sulphonamide treatment. Usually, if the drug is withdrawn at once the kidney recovers. Sulphadimidine rarely causes this complication.

Agranulocytosis (p. 154) and aplastic anæmia (p. 149) are very rare but grave complications. Drug rashes, drug fever, headache, nausea and depression are common but seldom serious.

Empyema.—Empyema means pus in the pleural cavity. The pleura may be attacked when inflammation spreads from the underlying lung, in pneumonia, bronchiectasis or lung abscess. Empyema may also be caused by penetrating wounds of the chest. Empyema complicating pneumonia is much less common than it was in pre-antibiotic days.

The formation of an empyema is suspected if the fever recurs after the temperature has fallen, or if the fever does not respond to treatment, especially if the patient is pale, breathless, sweaty and ill-looking. Mild cases may easily be missed for some time, or an empyema may only be discovered on X-ray. The diagnosis is clinched by "exploring the chest" with a needle, when some of the fluid can be drawn off with a syringe and examined.

Treatment depends on the kind of fluid which is found. If it is scanty, and looks yellowish and almost clear it probably contains few pus cells and no bacteria, and a little fluid of this sort will very likely clear up on its own, or after repeated aspiration. If the fluid is purulent as much as possible is withdrawn, using a large syringe and wide bore needle. A solution of penicillin (half to one million units) is then injected into the pleural cavity. (If the organism proves to be penicillin resistant another antibiotic must be used, but fortunately empyema is generally caused by penicillin sensitive organisms.) Often the pus is so thick that it is difficult to aspirate; *streptokinase* may then be injected as well as penicillin—it helps to liquefy the pus so that it no longer clogs the needle. (Streptokinase is a product of the haemolytic streptococcus; because it liquefies pus it enables streptococci in septic lesions to spread. In the treatment of empyema we make use of this liquefying action to help the patient instead of the streptococcus.)

Aspiration of pus and injection of penicillin and streptokinase is repeated daily or on alternate days, until the fluid withdrawn proves to be sterile. Regular aspiration may then be continued until very little fluid is left. The rest generally absorbs over the next three or four weeks and the underlying lung re-expands.

This treatment is successful in most cases. *Surgical drainage of the*

chest is now rarely necessary, and is reserved for those cases where the pus is inaccessible or too thick to be aspirated through a needle.

General Nursing Care and After Treatment.—In spite of the dramatic results of antibiotic treatment these patients still need most careful nursing, fresh air and plenty of good food to speed their recovery. Any septic process is apt to cause anæmia and wasting, so empyema patients need extra iron and protein-containing foods; vitamins, too, probably accelerate healing. (See Chapter 2.)

When the pleura is full of pus or fluid the lung underneath *collapses* and must be helped to re-expand, for collapsed lung cannot function properly and is also apt to become bronchiectatic (p. 104). *Breathing exercises* help to re-expand the lung once the pleura has been cleared of pus. These exercises may be directed by a physiotherapist. Nothing expands the lungs better than blowing on a wind instrument, and children may be encouraged to blow on toy trumpets as long as they can and until the noise becomes intolerable. After discharge from hospital empyema patients should have a long convalescence at the seaside or in the country.

Bronchopneumonia

This is much commoner than lobar pneumonia. Whereas lobar pneumonia often suddenly attacks a previously healthy person, bronchopneumonia generally complicates other illnesses—for instance chronic bronchitis or heart disease, or acute infections such as measles, whooping cough or influenza.

The *symptoms* of bronchopneumonia often come on gradually. An elderly man, who for years has suffered from bronchitis with cough and sputum, becomes ill and feverish, while the cough grows worse, the sputum more copious and purulent, and the breathing more laboured. In acute infections such as influenza, however, bronchopneumonia may develop rapidly, with cough, sputum, rapid breathing and a marked deterioration in the patient's general condition.

Staphylococcal pneumonia is a particularly dangerous form of the disease; it has caused a large proportion of the deaths from pneumonia during recent influenza epidemics. The illness is apt to be severe and prolonged. *Lung abscesses* are common in staphylococcal pneumonia. An abscess may rupture into the pleural cavity, causing *pyopneumothorax* (pus and air in the pleura).

Clinical and X-ray examinations show that patches of inflammation are scattered throughout both lungs—most often at the bases. The organisms causing the lung infection can be identified by culturing the sputum; the commonest are the *pneumococcus*, *Hæmophilus*

influenzæ (so-called because it was once thought to cause influenza) and *staphylococcus aureus*. Sensitivity tests will identify the antibiotics to which these organisms are sensitive.

Complications.—Pulmonary collapse (p. 101) is common in bronchopneumonia. *Lung abscess* may complicate pneumonia—especially staphylococcal pneumonia. *Bronchiectasis* may develop after bronchopneumonia in childhood—especially after repeated lung infections. *Empyema* is rare.

Treatment.—The *general treatment* is the same as for lobar pneumonia. The choice of *antibiotic* used will depend on the organism causing the infection. Penicillin or tetracycline are often given until the results of sensitivity tests are known, when it may be necessary to change to another drug to which the organism is more sensitive. In staphylococcal pneumonia the organism is often resistant to all the commoner antibiotics; erythromycin or novobiocin or both may then be used. These drugs may be used from the start in a severe, acute illness when staphylococcal pneumonia is suspected.

Treatment for pulmonary collapse (p. 102) will often be required.

Prognosis.—This is very variable; bronchopneumonia may be a mild illness which clears up rapidly after a short course of penicillin, or an overwhelming infection which is fatal within a few hours. Most of the deaths occur at the extremes of life, among infants and the aged. Staphylococcal pneumonia, however, has a high mortality at all ages.

Virus Pneumonia

This variety of pneumonia is less common than those so far described. Virus pneumonia usually starts as a feverish illness with sore throat and headache. A dry cough is common, and the patient may develop the usual symptoms and signs of bronchopneumonia. Often, however, chest symptoms are slight or absent, and the patient is sent into hospital as a "P.U.O." (pyrexia of unknown origin). The fever may last from a few days to several weeks. Hæmolytic anæmia (p. 150) develops in some cases, and the patient may become jaundiced.

Investigations.—The X-ray shows one or more patches of consolidation. The white count is usually normal, unlike the high counts of lobar and bronchopneumonia. Examination of the blood, sputum and throat washings may, in some cases, lead to the identification of the virus causing the infection. This is much more difficult than the identification of bacteria such as pneumococci or staphylococci. Viruses are too small to be visible under an ordinary microscope, and,

in addition, viruses will only grow in living cells, whereas bacteria can be cultured on agar jelly and other preparations. Virus identification usually takes several weeks, so is not of much help in the immediate diagnosis of virus pneumonia; this is made on clinical and X-ray findings.

Treatment.—Although most viruses are resistant to antibiotics, these drugs are generally prescribed in virus pneumonia, because bacteria often invade the lung already damaged by virus. (These bacteria are called *secondary invaders*). The general treatment is similar to that of bronchopneumonia.

Prognosis.—Patients with virus pneumonia almost invariably recover.

Psittacosis

This is mainly an infection of birds, such as parrots and budgerigars; occasionally diseased birds infect their human contacts. The disease is similar to virus pneumonia, but it is more severe and may be fatal.

Psittacosis is caused by a virus which is larger than most viruses and may respond to antibiotic treatment. Tetracycline is generally prescribed in psittacosis.

Q Fever*

This is an infection caused by rickettsia† (organisms between bacteria and viruses in size). It is probably less uncommon than was once thought. The symptoms are those of virus pneumonia; headache is particularly severe. *Tetracycline treatment* may be effective.

(2) Pulmonary Collapse (Atelectasis)

Wherever a bronchus becomes blocked, the air which is trapped in the lung beyond the blockage is absorbed and that part of the lung *collapses*. The bronchus may be blocked by thick secretions and sticky sputum in pneumonia. Or the blockage may be caused by an inhaled foreign body (p. 105). An enlarged gland at the root of the lung may compress a neighbouring bronchus; this sometimes happens in primary tuberculosis. The scarring which may follow bronchopneumonia may narrow or completely obstruct a bronchus.

* Originally called "Query Fever" before the cause was known—hence "Q fever".

† Named after Dr. Ricketts, who died of typhus while investigating this, the most important rickettsial disease.

Bronchial tumours (e.g. cancer) often obstruct a bronchus and cause collapse of the lung beyond.

A collapsed lobe of lung is very apt to become infected, as the blockage prevents drainage of secretions. Lung abscess or bronchiectasis may then develop.

Symptoms.—Collapse of small segments of lung causes no special symptoms, the diagnosis being made on X-ray. In other cases the symptoms are those of the primary disease—pneumonia, lung cancer etc. The symptoms of *massive collapse* are described below.

Treatment. This aims at *clearing the obstructed bronchus and preventing or curing infection.*

Sticky sputum is one of the commonest causes of bronchial obstruction. If sticky sputum can be liquified the patient can often cough it up himself. *Steam inhalations* help to liquify sticky sputum, as may inhalations of special mucus-liquifying preparations. *Percussion postural drainage* helps the patient to cough up his sputum; the patient's position is such that the blocked bronchus is uppermost, while the physiotherapist taps, knocks or pummels the chest wall to help to dislodge impacted secretions.

Foreign bodies can usually be removed at bronchoscopy, and the bronchoscope may also be used to free the obstruction caused by sticky sputum.

Since bronchial obstruction and pulmonary collapse are so often associated with lung infection, antibiotics are necessary.

Massive Collapse of the Lung

This occasionally happens a day or two after an operation—often an upper abdominal operation such as gastrectomy. The patient suddenly becomes blue, breathless and distressed; he may become unconscious. The pulse and respiration rates are high and the temperature is raised. Clinical examination and X-rays show that the whole of one lung has collapsed. (See Fig. 26, though in this case the collapse was caused by a growth). The *cause* of massive collapse may be a tough plug of mucus, which the patient cannot cough up because of pains at the site of operation.

Treatment. The patient's position should be changed frequently and he should be encouraged to cough. He is given oxygen, and antibiotics are prescribed as required. The obstruction can sometimes be relieved at bronchoscopy.

Prevention.—Patient's recovering from major abdominal operations should not be immobile for long periods. Nurses should help

them to change position at frequent intervals. Breathing exercises may be helpful. Large doses of morphia depress respiration and inhibit coughing, and so may pre-dispose to collapse of the lung. Pethidine and anidone have a less depressing effect on respiration and are often preferred when a pain reliever is required. Skilful surgery, anæsthesia and nursing together make post-operative massive collapse a rare occurrence.



Fig 26 (a)

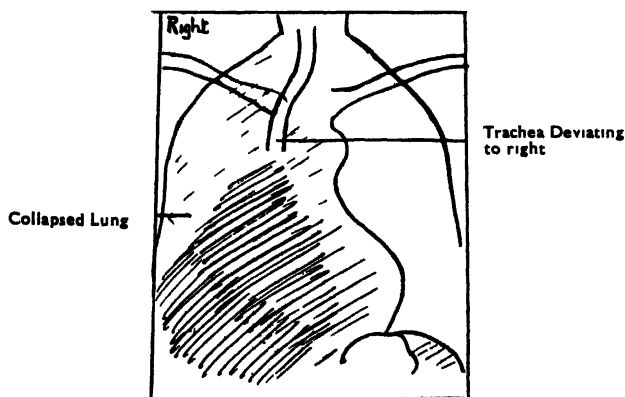


Fig 26 (b)

Massive collapse of Right lung due to Carcinoma of the Right Main Bronchus

(3) Lung Abscess

This is a localised collection of pus in the lung. An abscess may develop because *inflamed lung tissue breaks down*, or because a *bronchus is blocked* and septic material is dammed back in the lung beyond the blockage. Lung cancer is one of the commonest causes of bronchial obstruction and of lung abscess: or the cancer itself may break down and form an abscess. A lung abscess is sometimes caused by the *inhalation of septic material*—e.g. during operations on the nose or throat. Or the infection may be brought by the blood stream, as in staphylococcal septicæmia; the lungs may be riddled with abscesses in such cases. Finally, severe *chest injuries* can cause lung abscess.

Symptoms.—Cough, fever, and pain in the chest occur, as in pneumonia. But the “pneumonia” does not respond to treatment as expected. Fever persists, the patient looks ill, and after a few days begins to bring up *purulent sputum*. Should the abscess remain untreated the patient goes downhill, loses weight, becomes anæmic and clubbing of the fingers develops unless he coughs up the abscess contents, so that the cavity can close. *Empyema* is a fairly common complication.

Investigations.—The *sputum* is cultured, and organisms tested for sensitivity to antibiotics. On *X-ray* the abscess shows up as a cavity surrounded by consolidated or collapsed lung. *Bronchoscopy* is often necessary if there is a bronchial block. *Blood counts* show a raised white count and, often, anæmia.

Treatment.—Intensive *antibiotic treatment* is essential; the choice of antibiotic depends on the results of sputum tests. *Postural drainage* and *physiotherapy* will help the patient to cough up the pus in the abscess. When an abscess follows obstruction of a bronchus by a foreign body, the latter can often be removed at *bronchoscopy*. *Operation* is necessary if the abscess persists in spite of medical treatment, the affected lobe or segment being resected.

Prognosis.—The majority of patients with lung abscess recover completely if the diagnosis is made early and intensive treatment started promptly.

(4) Bronchiectasis

To the doctors and nurses of a former generation the word “bronchiectasis” implied a wasted patient with blue lips and

clubbed fingers, who suffered from a racking cough and blood spitting, and who filled the ward with the reek of his copious stinking sputum until his inevitable death. Nowadays, modern X-ray techniques have enabled us to diagnose bronchiectasis in the early stages of the disease, antibiotics keep infection under control and the chest surgeons can in some cases effect a complete cure.

A patient with advanced bronchiectasis, as described above, is still seen from time to time. More often the condition is discovered in one who is admitted for chest investigation who is often not desperately ill. Such a patient may be a child or adult, more often male than female and, as in most chest infections, is more often

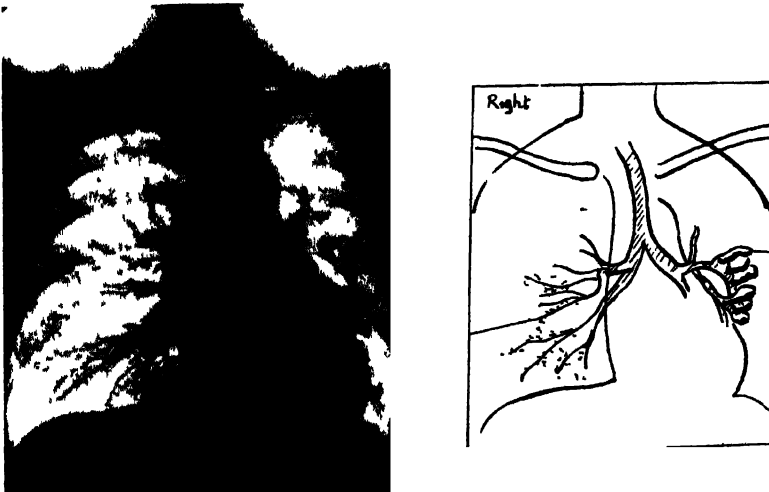


Fig. 27.

Bronchogram showing gross disease on left normal bronchi on right (see p 108), Dr Thomas's case

badly off than well-to-do. He generally has a story of a chronic cough, perhaps dating from measles or whooping cough or from a recent attack of pneumonia. Other bronchiectatics have "always had a winter cough". In some cases the patient may have coughed ever since "something went down the windpipe"—perhaps a peanut or part of a small child's toy. The cough may have lasted for years without incapacitating him; he may or may not bring up

much sputum. Sometimes the first symptom to bring the patient under medical care is a hæmoptysis.

In an early case of bronchiectasis observation of the patient may reveal little that is abnormal. A good many of them, however, have clubbed fingers. There may be purulent, greenish yellow sputum which in late stages becomes copious and offensive. Advanced cases may bring up a pint or more in the 24 hours.

The first investigation necessary in any case of chronic cough or hæmoptysis is an *X-ray of chest*. This may show only minor abnormalities in bronchiectasis. A history suggestive of the latter then calls for a *bronchogram*. An oil, such as lipiodol, which shows up in the X-ray, is run down the trachea either through a tube passed into the larynx or by injection directly into the trachea through the skin of the neck. An anæsthetic is necessary for children. The oil runs down into the bronchi, whose shape and arrangement can then be seen in the X-ray.

In bronchiectasis the bronchogram shows that one or more bronchi are dilated. The dilated bronchi are usually those of one lower lobe, but other lobes and both lungs may be affected.

If the presence of an inhaled peanut or other obstruction is suspected a bronchoscope may be passed, through which the bronchi can be seen and any obstruction noted.

The *sputum* is always searched for tubercle bacilli, for the same patient may have both bronchiectasis and tuberculosis.

Causes of bronchiectasis.—Dilated bronchi may be produced in various ways. The bronchial walls may bulge because they are weakened by *chronic inflammation* as in bronchitis. *Constant coughing* may distend them. The bronchi may be pulled out of shape by the *fibrous tissue* formed in the scars of old patches of tuberculosis or pneumonia, either of which may also have infected and weakened the bronchial wall. Or bronchiectasis may be the result of *collapse* of part of the lung. If a child inhales a bead into a bronchus the air in the lung beyond the block is gradually absorbed and the airless lung collapses. The bronchi in the collapsed lung expand because they are no longer supported by the air cushion of aerated lung. Pulmonary collapse and bronchiectasis may also occur when a bronchus is blocked by a tough plug of mucus or by a tumour or by enlarged glands at the root of the lung. Rare cases of *congenital bronchiectasis* occur.

Dilated bronchi, whatever the cause of their dilation, readily become infected, especially if their walls are already weakened by inflammation. A large variety of organisms flourish in the secretions

which accumulate in the bulges and which are eventually coughed up as sputum. The inflammation may spread and involve a blood vessel, resulting in hæmoptysis. The long continued sepsis will eventually cause fever and loss of weight if unchecked.

Treatment.—This may include attempts to *drain* the infected bronchi, to *disinfect* them or to *remove* the affected part of the lung. In any case the patient's general condition must be sustained in every possible way with good food, fresh air and good nursing.



Fig. 28 A child with bronchiectasis undergoing postural drainage in a special adjustable bed

If a bronchus is blocked by a foreign body this can often be removed by means of the bronchoscope; there is then a very good chance of re-expanding the lung and curing the bronchiectasis. Breathing exercises are helpful in these cases.

Draining the Bronchi.—The patient lies so that the affected bronchi are uppermost. Postural drainage of the *lower lobes* is achieved by raising the foot of the bed on 18 inch blocks; the patient lies on his face, back or side according to the part of the lobe affected. In bronchiectasis of the *middle lobe* a twelve inch block is used; the patient lies with a firm pillow under the back, inclined slightly to

one side with the affected lung uppermost. Bronchiectasis of the *upper lobes* is uncommon; these lobes drain adequately when the patient is sitting up. Postural drainage, maintained for hours a day, enables the patient to bring up several ounces of sputum daily.

Disinfecting the Bronchi.—A course of penicillin or other antibiotic will usually reduce the amount of sputum, at least temporarily; long-term antibiotic treatment is sometimes advised. Intensive chemotherapy is necessary for the acute respiratory infections so common in bronchiectasis.

Operation.—Lobectomy—removal of one or more lobes of lung—may be considered if the patient has troublesome symptoms, if his disease is not too extensive and if he is fit to submit to a major operation. The results are excellent in selected cases. Only a small proportion of bronchiectatics, however, are suitable for operation, either because the disease is too slight to warrant it or too extensive to be operable.

Prognosis. This is much better than was once thought. In a recent series patients had already had their symptoms for an average of eighteen years at the time of the survey. Regular postural drainage, breathing exercises, and antibiotics when necessary will keep the majority in reasonable health for many years. Most are able to work or attend school. In a few cases, however, the disease is disabling. When death occurs it is usually the result of extensive lung sepsis or of secondary heart failure.

Case History.—J.O., aged 38, invalided from the Army after 16 years' service. He had been breathless for 7 years, and had had a cough and sputum a little longer. After his discharge from the Army he became increasingly breathless and found it hard to keep a job. He had a cough and copious sputum: he was cyanosed and had severe clubbing of the fingers. Bronchogram (Fig. 27) showed gross distortion and dilatation of bronchi on the left; those on the right were normal. His condition deteriorated, and surgery was advised. At operation the whole of the left lung was found to be diseased and was removed. This cured his cough and sputum, but he was still very breathless.

(5) Tuberculosis

Tuberculosis is one of the commonest infectious diseases. It is believed to cause some 3-5 million deaths a year all over the world. It may affect almost any part of the body. Most of this chapter is devoted to tuberculosis of the lungs—by far the most important form of tuberculosis. First, however, comes a brief account of how the infection is spread and how it develops in the body.

The tubercle bacilli, which cause tuberculosis, are spread by tuberculous patients, and, to a much smaller extent, by tuberculous cows. *Bovine tuberculosis*, as it is called, is caused by drinking infected, unpasteurized milk. It may cause abdominal tuberculosis (p. 182), tuberculous cervical glands (p. 161) and other forms of non-pulmonary tuberculosis.

Tuberculous infection much more often comes from a human source, nearly always a patient with tuberculosis of the lungs whose sputum contains tubercle bacilli (known as an *open case*). The bacilli are expelled into the air when he coughs. Other members of the household are likely to inhale these organisms, or (much less often) to swallow them by eating contaminated food. Infection may also spread to people he meets at work, or in buses or shops. Direct contact is not necessary, for tubercle bacilli survive some time outside the body if they are kept moist (they die rapidly if dried or exposed to sunlight).

The Primary Infection

The first entry of tubercle bacilli into a man, woman or child constitutes the *primary infection*. Most commonly the bacilli lodge in the lungs and the real little patch of tuberculous inflammatory tissue develops—the *primary focus*. Fortunately, most of us have sufficiently high resistance to overcome this primary infection; we do not become ill and even our chest X-rays may be clear. The majority of people in this country over the age of 30 have had their primary infection, usually without ever being aware of it.

Tuberculin Tests.—These enable us to identify *those who have ever been infected* with tubercle bacilli. A small quantity of tuberculin—an extract of tubercle bacilli—is injected into the skin. If within a few days the skin around the site of injection becomes red and swollen the test is positive; this shows that the person has been infected *at some time*—it may be weeks, months or years ago. It does *not* mean that he is suffering from tuberculous disease.

In the *Mantoux test* 0.1 ml. of a tuberculin solution (1/100, 1/1000 or 1/10,000) is injected *intradermally*, so that the solution raises a little button-like wheal. Mantoux tests are usually done by doctors, but the nurses generally do the *Heaf test*. For this test a drop of tuberculin solution is placed upon the cleansed skin of the forearm, and the *multiple puncture gun* applied. This produces six minute punctures through which the tuberculin solution penetrates the skin.

Primary Tuberculosis

When the primary infection causes an illness, or changes in the lung which can be seen on X-ray, the condition is called *primary*

tuberculosis. The patient is usually a child or young adult. Symptoms, if any, are vague—general ill-health, fatigue, loss of weight, a low fever. Often the diagnosis is made on X-ray of an apparently healthy contact of a tuberculous patient. The primary focus may show up on X-ray as a small patch, and the glands at the root of the lung are often

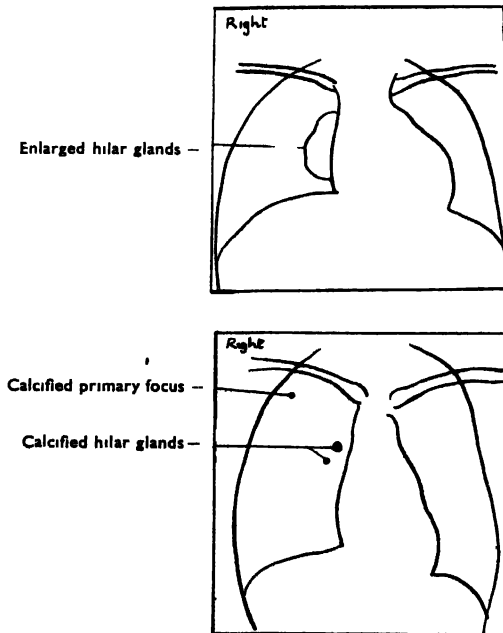
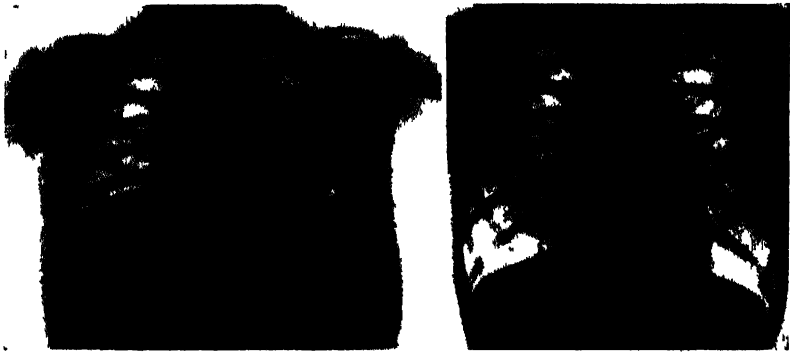


Fig. 29. Primary Tuberculosis.

A. Girl aged 20 months. X-rayed as contact of father. No symptoms, X-rays show enlarged hilar glands on the right. B. Same child aged 7. Hilar glands now calcified; a calcified primary focus is also seen in the right upper zone, though no lung focus was visible in the first film. This child was followed up throughout childhood; she had no symptoms at any time. No treatment was given (this was in pre-chemotherapy days). (Dr. Thomas's case.)

enlarged, especially in children. The primary focus together with the enlarged glands are together known as the *primary complex*. The tuberculin test is strongly positive.

Erythema nodosum (p. 279) may lead to the diagnosis of primary tuberculosis. It may occur soon after the primary infection in older children and adults. Although this rash may occur in other conditions its appearance always means that the patient must be investigated for tuberculosis.

Treatment.—If the X-ray suggests active primary tuberculosis the patient is usually given anti-tuberculous drugs (p. 117) even in the absence of symptoms.

The younger the patient the more urgent the need for treatment.—Babies under the age of two have so little resistance to tuberculosis. and the risks of miliary tuberculosis (see below) and tuberculous meningitis (p. 326) are so grave that infants with a positive skin test are given antituberculous drugs, even if the X-ray is normal.

Some patients with primary tuberculosis will require admission to hospital for a period of weeks or months. All will need prolonged follow-up at the Chest Clinic. *Contacts must be X-rayed too*, and unsuspected infectious cases may be discovered in his way.

Case History.—G.C., aged 12, had been "out of sorts" for some months. She then developed erythema nodosum. Tuberculin test strongly positive. X-ray showed primary tuberculosis. Her family were X-rayed at the chest clinic; none had symptoms of chest disease. Both parents had active pulmonary tuberculosis; the father's sputum was positive—he had evidently infected both wife and daughter. The other two children had negative tuberculin tests and normal X-rays; they were given B.G.C. (p. 120). They were looked after by an aunt while both parents were treated in hospital.

Course and Prognosis of Primary Tuberculosis

Even in the absence of treatment, the *primary complex generally heals with few or no symptoms*. The X-ray may clear completely, or primary focus and hilar glands may become impregnated with chalky deposits which show up clearly in the film. These *calcified lesions* are often seen in X-rays of perfectly healthy people.

In a minority of cases the outcome is less happy. The infection may spread to the lung surrounding the primary focus, developing into the adult type of pulmonary tuberculosis. Or the pleura may be involved, resulting in pleural effusion (see below). Or the enlarged glands may press upon a bronchus, causing *collapse* of part of the lung supplied by that bronchus (p. 101). Or a tuberculous gland

may rupture into a bronchus, spreading the infection widely into the lung beyond (*tuberculous pneumonia*). In some cases tubercle bacilli invade the blood stream: an acute, overwhelming blood stream infection causes miliary tuberculosis (see below), whereas a less severe and more chronic infection may result in tuberculosis of the bones, joints, kidneys, etc.

The outcome of the primary infection depends upon the *size of the dose of infecting bacilli* and upon the person's *resistance*. Resistance depends upon the general health and nutrition, on age (babies have little resistance to tuberculosis), on race (for instance, the Irish are more susceptible to tuberculosis than the English) and on many other factors which we do not understand.

Miliary Tuberculosis

This is a form of tuberculous septicæmia. It is commonest among infants and young children exposed to a heavy dose of infection—for instance, the patient may be the child of a man with unsuspected infectious pulmonary tuberculosis. Miliary tuberculosis usually occurs within a few months of the primary infection; it is an uncommon complication of the later stages of the disease. It is much less common today than formerly.

The onset of miliary tuberculosis may be rapid or gradual. The patient becomes ill and feverish; he sweats profusely and rapidly loses weight. In the later stages he becomes breathless and cyanosed; often there is little or no cough. Tuberculous meningitis (p. 326) may develop at any stage.

The chest X-ray shows a "snow storm" effect which is characteristic (Fig. 30). The little white patches represent *tubercles*—little patches of tuberculous inflammation. In fatal cases, post mortem examination shows that these tubercles are widely scattered throughout the body. The tubercles are about the size of grains of *millet* (bird seed)—hence the name.

Treatment.—Full doses of anti-tuberculous drugs (see below) are given for at least a year. If the diagnosis is made quickly and treatment given promptly complete recovery is the rule. If meningitis develops the outlook is more serious, especially in infants. Both these diseases were invariably fatal in pre-chemotherapy days.

Case History.—M.L., aged 4 months. Losing weight for a month. X-rayed as contact of grandfather who had just been found to have tuberculosis. Miliary tuberculosis found (Fig. 30). Tubercle bacilli found on gastric lavage. Complete recovery with chemotherapy. X-rays showed rapidly clearing.

Pleural Effusion

This is usually a form of *post primary tuberculosis*, which may occur within 6 months of the primary infection. The onset of the illness may be gradual or sudden, with fever, pain in the chest and breath-



Fig 30 Miliary Tuberculosis

Baby of 4 months (see page 112) The mottling is rather coarser than is usual for miliary tuberculosis (Dr. Springett's case)

lessness. X-ray of the chest shows that there is a collection of fluid in the pleural cavity. When the fluid is aspirated it proves to be *clear* and *straw coloured*. The tuberculin test is strongly positive. Tubercle bacilli can seldom be found either in the fluid or the sputum (if any), but a pleural effusion of this type is always considered to be tuberculous unless proved otherwise. This is because, in pre-chemotherapy days, about 30 per cent. of these patients later developed frank pulmonary tuberculosis.

Treatment.—The patient is admitted to hospital and is kept in bed for some weeks. A small quantity of fluid is withdrawn for examination—often no further aspirations are necessary. Chemotherapy is given for 6–12 months. Steroids (see below) may also be given and may hasten the absorption of the fluid and the disappearance of symptoms. After, say, 6 weeks in bed the patient is allowed up for increasing periods and is discharged from hospital after about three months. He is likely to be back at work in six months. He attends the Chest Clinic for follow-up for several years. Pulmonary tuberculosis very rarely develops after an efficiently treated pleural effusion.

Tuberculosis of the Lungs (Phthisis, Consumption)

We have already seen that the primary focus may spread and involve other parts of the lung. Tuberculosis of the lungs may also be caused by the re-infection of a person who had his primary infection perhaps many years before.

Pulmonary tuberculosis still causes a great deal of illness, though it is nothing like as common or as deadly as it was only a few years ago. The death rate has been falling for over a hundred years (apart from set-backs during the two world wars) and it is falling still. Tuberculosis of the lungs caused 3,094 deaths in 1960—less than the number of road deaths—compared with 14,079 in 1950. In 1900 the disease caused 1,330 deaths per million of the population; in 1940, 590; in 1958, 89. At first this great improvement was mainly due to improvement in the social conditions. Since the introduction of chemotherapy the mortality has fallen still more rapidly.

The Tuberculous Patient

The patient with pulmonary tuberculosis may be male or female, young or old, rich or poor. The disease is, however, commoner among men than women, among poor than rich. Among women tuberculosis is commonest before the age of 30; male patients are

more often middle aged or elderly, and *most of the deaths now occur among these elderly men*. Romantic young poets and lovesick maidens no longer die of consumption.

Symptoms.—*In the early stages there are none*. Many cases of pulmonary tuberculosis are now diagnosed following a chest X-ray of an apparently healthy person.

Those who have symptoms most often have a *cough*. At first it is dry; later the patient may bring up a little yellowish *sputum*. *Hæmoptysis* is fairly common. The patient may *lose weight* and *feel irritable, tired, ill and feverish*. In women patients there may be *menstrual irregularities*. Even those who deny all symptoms often realize, after treatment, that they are now feeling much better and stronger than they were before.

Older patients, who have probably had the disease for many years, and, often, bronchitis as well, finally become severely breathless and blue and symptoms of heart failure develop. The nurse working in a tuberculosis ward may therefore have under her care cases of all degrees of severity, from fit young men, annoyed at being in bed, to old men dying of respiratory and cardiac failure.

Investigations.—*Chest X-rays* are essential both for diagnosis and in the assessment of progress. The “fit” young man mentioned above probably has a small area of fluffy mottling near the top of one lung. In a more severe case the mottling is more extensive and there may be a *cavity*—showing as a ring-shaped shadow, and formed as a result of the breakdown of dead infected lung tissue. The lungs of the dying old man may be riddled with cavities and also scarred with fibrous tissue, showing up on X-ray as dense streaky shadowing obliterating much of his lung and distorting normal structures.

Tomography.—A special X-ray apparatus is used which can be focused at any desired depth from the surface; the tissues in all other planes are thrown out of focus. A cavity which could not be seen in the straight film may show up in the tomogram, and it can also be accurately located. Tomography is particularly useful when surgery is contemplated.

Sputum examination.—At least three specimens of sputum are examined in any suspected case. If tubercle bacilli are very numerous they may be seen on examining the stained film under the microscope; otherwise cultures are necessary. (Unfortunately the tubercle bacillus is a slow grower, and the results of cultures are not known for at least six weeks.) In either case a positive result means, firstly, that the patient *certainly has active tuberculosis*, and, secondly, that he is *infectious*. If there is no sputum, *laryngeal swabs* are taken. Some-

times *stomach washings* may be examined—especially in children, who usually swallow their sputum.

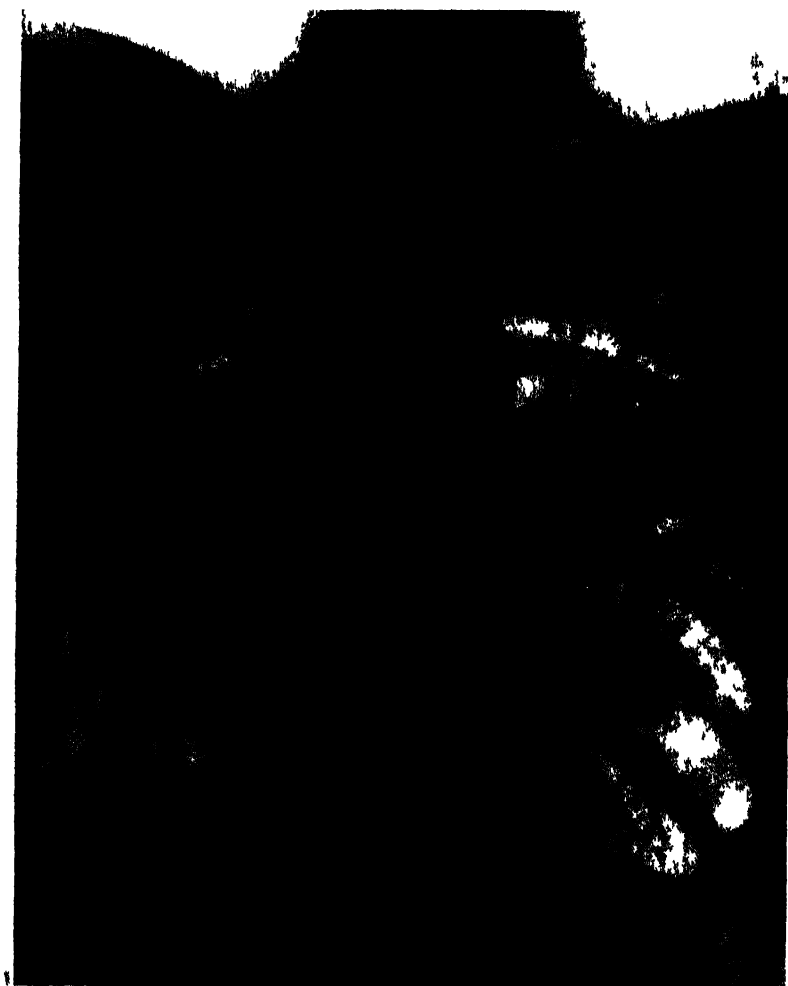


Fig 31 Pulmonary Tuberculosis

Widespread patchy infiltration in both lungs with a large cavity just below right clavicle (See p 122)
(Dr Luntz's case)

Treatment.—The treatment of pulmonary tuberculosis is lengthy, and calls for the co-operation of physicians, surgeons, nurses, health visitors and, not least, the patient and his family. These are the main forms of treatment:

(1) Rest in bed: to improve the general condition and promote healing.

(2) Anti-tuberculous drugs (chemotherapy): to eradicate tubercle bacilli.

(3) Steroids: to relieve toxic symptoms.

(4) Collapse therapy: to restrict movements of the affected lung and help to close cavities.

(5) Resection: to remove diseased lung tissue at operation.

Chemotherapy and rest are the essentials; steroids and surgery are valuable in some cases.

(1) *Rest*.—At least three months' bed rest are usually advised in active pulmonary tuberculosis. The best results are obtained if the patient is admitted to hospital and has skilled nursing care. As there is now no shortage of beds, he can usually be admitted straight away. Treatment can be given at home if necessary, but this is not usually recommended.

After three months during which the patient gets up only to the toilet, if all goes well he is allowed up for increasing periods. He can go home when he is up for several hours a day—say after 6 months in hospital. In mild cases his stay in hospital may be much shorter.

(2) *Chemotherapy*.—The discovery of streptomycin in by Dr. Waksman and his colleagues in the U.S.A. opened a new era in the treatment of tuberculosis; since then this once intractable condition has become one of the most curable of chronic infections. At present the three main drugs used are streptomycin, P.A.S. (para-aminosalicylic acid) and isoniazid (iso-nicotinic acid hydrazide). At least two drugs are always used together, often all three. This is because tubercle bacilli rapidly become resistant to any one drug used alone; this would mean that that drug would in future be useless in the treatment of that patient or of any other person he happened to infect.

Streptomycin is given by intramuscular injection (1 gramme daily)—usually for the first few months of treatment, while the patient is in hospital. It is apt to cause dermatitis in those who handle it without special precautions—*wear a mask and rubber gloves while giving injections*.

Streptomycin may cause fever and rashes during the first two or three weeks of treatment—less often in the later stages. The symptoms clear up if the drug is stopped. The patient may then be desensitised by giving a minute quantity of the drug and gradually increasing the dose. Toxic symptoms which are much more serious

—though fortunately uncommon—are *unsteadiness* and *deafness*; visual disturbance is another danger signal. If you notice a patient swaying or staggering down the ward, or becoming hard of hearing, *inform the doctor immediately*; he will probably stop the streptomycin. Even a few more doses may cause irreparable damage to the *vestibular nerve* (of balance) or the *auditory nerve* (of hearing).

P.A.S.—10-20 grammes daily are given by mouth in divided doses, in the form of cachets, tablets or liquid preparation. Drug rashes and fever are common in the early stages of treatment, and nausea and digestive upsets may occur at any stage. *Jaundice* is a rare but serious complication.

Isoniazid.—The dose is 200-300 mgm. a day by mouth. Combined cachets of *P.A.S.* and isoniazid are usually prescribed for patients taking the two drugs. Toxic symptoms are rare.

A typical course of treatment is as follows:

(a) *All three drugs for three months* (or until the pathologist reports on the drug sensitivity of the tubercle bacilli in the sputum).

(b) If the bacilli are sensitive to all three drugs, *P.A.S.* is then discontinued (it is the most unpleasant to take) and the patient receives streptomycin and isoniazid for a further three months, or till he leaves hospital.

(c) After discharge from hospital he takes *P.A.S. and isoniazid* in the form of combined cachets, till he has had *two years' total chemotherapy*.

(3) *Steroids.*—Ill patients with much fever get better more quickly if steroids are given as well as anti-tuberculous drugs—the temperature falls, appetite improves and weight increases. We have already seen (p. 60) that the lighting up of latent tubercle is one of the known complications of steroid treatment, so it may seem strange to give these drugs in active tuberculosis. The reason is that the *infection* is, in treated cases, *controlled by the anti-tuberculous drugs*, while steroids reduce symptoms of *inflammation and toxæmia*. Pleural effusion (an inflammatory exudate) is absorbed more quickly when steroids are given (see above). Steroids also help to control drug reactions.

(4) *Collapse Therapy.*—All forms of collapse are much less used today than formerly. As a rule, lungs heal and cavities close after some months of rest and chemotherapy. But in very chronic cases cavities may be kept open by tough fibrous tissue, or tubercle bacilli may be drug resistant. By partially collapsing the lung we limit its movements and allow cavities to close.

Thoracoplasty is an operation which produces a *permanent degree*

of collapse by removing parts of several ribs over the diseased lung, so allowing the chest wall to fall in. It is usually done in two stages. All other forms of collapse therapy are now rare.

Air may be introduced into the pleural cavity (*artificial pneumothorax*) or the peritoneal cavity (*pneumoperitoneum*), thus partially collapsing and restricting the movements of the adjacent lung. These forms of treatment are hardly ever started nowadays, but nurses will probably see the occasional patient coming up for refills of his "A.P." or "P.P." which he has had for years.

(5) *Resection*.—Here the diseased part of the lung is removed at operation. It is most successful when the disease remaining after adequate chemotherapy is limited to part of one lung.

After Care and Follow Up

After he leaves hospital the patient usually rests at home for a time before returning to work. He is looked after by the family doctor and, if necessary, the District Nurse—though by this stage he is usually up most of the day and is not having streptomycin injections. At regular intervals he will be examined and X-rayed at the Chest Clinic—at first every two or three months, later, once or twice a year. *Yearly examinations are continued indefinitely* because relapses do occur, though they are uncommon after adequate chemotherapy. Defaulters are visited by the Tuberculosis Health Visitors, who use their gifts of persuasion in getting them up to the Clinic.

After a period of rest at home the patient is usually fit for work—say within a year of diagnosis. If his former job was heavy and he is only fit for light work he may be registered as a *disabled person*. All large firms are bound by law to employ a certain percentage of disabled workers. The Disablement Rehabilitation Officer at the Labour Exchange helps the disabled to find suitable work.

Prognosis.—*The vast majority of adequately treated patients recover from pulmonary tuberculosis.* Remember this when nursing tuberculous patients, who are apt to be appalled when they first hear the diagnosis. They require repeated reassurance and encouragement. In most cases the patient will be back at work within a year; women are usually fit for that most arduous job—marriage and childbearing.

The X-ray usually does not clear completely, but most patients become symptom free and *all but a few are rendered sputum negative*.

Treatment is least successful with elderly men with grossly diseased and distorted lungs. Even here the sputum usually becomes negative. Chemotherapy may have to be continued indefinitely.

Prevention of Tuberculosis

Tuberculosis spreads when *infection* reaches *susceptible subjects*. Prevention, therefore, involves *reducing the chances of infection* and *increasing resistance*.

Reducing Infection.—Drug treatment not only cures patients—it prevents the disease spreading to others because the sputum so rapidly becomes negative.

The most dangerous source of infection is not the known case but the undiagnosed one—he goes about unwittingly spreading tubercle bacilli. Therefore we must find these undiagnosed and, often, symptomless cases, so that they may be treated—both for their own sake and others’.

This means taking chest X-rays of large numbers of apparently healthy people, especially those who are frequently exposed to infection (contacts of cases, nurses) and those whose jobs involve a high risk of tuberculosis (seamen, miners). Much of this work is done at the Chest Clinic. Mass Radiography Units also X-ray many thousands of people a year—healthy volunteers and people with symptoms referred by their doctors; many cases of tuberculosis are found in this way.

The chances of infection are increased by *overcrowding*; housing problems are therefore important. The Tuberculosis Health Visitor visits the families of patients and reports on their housing. When there is gross overcrowding the Chest Physician can recommend re-housing.

Personal hygiene is important. The Health Visitor advises the patient about sputum disposal.

Prevention by Increased Resistance.—*B.C.G.* (Bacille Calmette Guérin). This is a vaccine consisting of *live* but *non-virulent* tubercle bacilli. It raises the resistance to infection with *virulent* organisms. A tuberculin test is done before giving B.C.G.; a positive result means that the person has already had a dose of ordinary tubercle bacilli so that B.C.G. would be valueless. B.C.G. is now offered to all tuberculin negative *contacts of patients, school leavers, and nurses*.

General resistance. Good health helps resistance; good health needs food; food needs money. The ordinary National Health Insurance Benefit does not go far when the breadwinner is off work for a year. Patients undergoing treatment for tuberculosis are eligible for special *tuberculosis allowances*. This helps the families and en-

courages the patients not to discharge themselves from hospital and go back to work too soon.

Prevention of Bovine Tuberculosis

Pasteurisation of Milk. Heat treatment kills most of the disease-producing bacteria in milk, including tubercle bacilli. *Pasteurised milk is therefore safe*, even if it comes from tuberculous cows.

Obviously it is better to have healthy cows. During the past 20 years tuberculosis has been eradicated from the herds of whole counties. "T.T." (tuberculin tested) milk comes from herds every member of which is tuberculin tested twice a year. Pasteurisation and the eradication of tuberculosis among cows has made bovine tuberculosis increasingly rare.

Summary of Preventive Measures: The Work of the Chest Clinic

The prevention of tuberculosis involves the co-operation of many different agencies, including Public Health (B.C.G.), Mass Radiography, the Ministries of Labour and Pensions (jobs and allowances) and the Ministry of Agriculture (bovine tuberculosis), and the Housing Authorities. The Chest Physician and the staff of the Chest Clinic are responsible for much of the preventive work. As soon as a case of tuberculosis is notified to the Medical Officer of Health (see p. 316) the Chest Physician is informed. He is usually responsible for the patient's treatment. The Tuberculosis Health Visitor is also notified, and visits the patient's home as soon as possible. She investigates the social and domestic situation, arranges for the examination of contacts at the Chest Clinic, and often does the Heaf testing and makes arrangements for B.C.G. vaccination. She also visits defaulters and persuades them to go up to the Clinic for follow-up. The Chest Physician and his staff can often help the patient over problems of jobs and housing, even if the final decisions are made elsewhere.

Conclusion

Three hundred years ago John Bunyan described tuberculosis as "the Captain of the Men of Death". This is still true in many parts of the world. Here we are more fortunate, and there is a reasonable hope that the disease will be virtually eliminated within the next generation. This will need unremitting perseverance in case finding, thorough treatment and follow up of cases, contact examination and the use of B.C.G. Much remains to be done before tuberculosis in

Britain is eradicated as completely as cholera was nearly a century ago.

(1) Miss B.B., aged 20, shop assistant, had a cough for two months and felt tired. She was referred by her doctor to the chest clinic and widespread disease of both lungs found (Fig. 31); there was an enormous cavity in the right lung, and a smaller one on the left. The sputum was positive. She was admitted to hospital, where she had streptomycin, P.A.S. and I.N.A. for 3 months, streptomycin and I.N.A. for another 3 months. On discharge she had P.A.S. and I.N.A. at home for 18 months. She returned to work within a year of diagnosis. After 18 months' chemotherapy this extensive disease had practically cleared, leaving only a small scar on the right.

(2) Mr. A. M., aged 48, a sheet metal worker. Referred to Chest Clinic after he had had a cough and sputum for 2 years; he had lost a little weight—could not say how much. *X-ray* showed widespread mottling, with a large cavity below right clavicle. Sputum positive on smear. Admitted to hospital forthwith. Discharged after 6 months; continued chemotherapy at home. After 18 months' drugs the mottled shadows had all cleared, but the cavity had not closed. He was re-admitted and the diseased part of the lung resected. He made an excellent recovery. In all he had 2½ years' chemotherapy.

(6) Cancer of the Lung and Bronchi

This is a common and highly fatal condition. Every year it causes more deaths, especially among men (five times as many men die of the disease as women). One male death in eighteen is now due to lung cancer.

Whereas the death rate from other cancers has altered little, the lung cancer death rate is rising by leaps and bounds. There were 6,568 lung cancer deaths in 1944, 21,992 in 1960. *It is very much commoner among cigarette smokers than non-smokers*; we shall return to this point later. It is also commoner in the large towns than in the country.

The lung cancer patient is usually a middle aged or elderly man with nicotine stained fingers. He has probably had a "smoker's cough" for years; recently he has consulted the doctor because the cough has got worse, or because of pain in the chest, blood spitting, or loss of weight. At this stage he may look quite well. Weeks or months later he will probably be wasted, breathless, and pale or cyanosed. There may be clubbing of the fingers.

Finally, he may suffer from a great variety of symptoms—swelling of the arm or face, hoarseness, difficulty in swallowing, headache, fits, severe back pain or paralysis of the legs, to name but a few. Occa-

sionally one of these symptoms first brings him to the doctor, chest symptoms being inconspicuous.

Investigations

(1) *X-ray*.—The diagnosis may be obvious from the first X-ray, or the appearances may be indistinguishable from those of tubercle or pneumonia.

(2) *Bronchoscopy*.—This is necessary whenever the diagnosis is in doubt. The growth may be seen through the bronchoscope, and a small piece may be taken for microscopic examination (*biopsy*). This clinches the diagnosis and may also suggest the best form of treatment.

3. *Sputum examination*.—If malignant cells are found in the sputum this also clinches the diagnosis. Unfortunately this test is seldom positive even in proved cases of lung cancer.

Explanation of the Symptoms.—A cancer is a *malignant growth*—that is, a mass of cells which divide and multiply without rhyme or reason, pressing on and destroying the surrounding healthy tissues. Malignant cells may also spread via the lymphatics to the local *lymph glands* and via the blood stream to *any part of the body*.

Lung cancers are particularly liable to spread in these ways, and this accounts for the variety of symptoms they produce. The *primary growth* may cause cough, sputum and hæmoptysis. *Pressure on nerves* and *destruction of ribs* causes pain in the chest. *Pressure on the great veins and lymphatics* causes swelling of arms and face. *Pressure on the nerve to the larynx* causes hoarseness. *Pressure on the œsophagus* causes difficulty in swallowing. *Secondary growths in the brain*—which are fairly common—cause headache and many other symptoms (see p. 299). *Secondary growths in the vertebræ*—also common—cause severe pain in the back and, often, paralysis through damage to the spinal cord. *Loss of weight* and *anæmia* are common to all advanced cancers; the reasons for this are not fully understood.

Treatment.—If investigations show that the growth is operable, and the patient is fit for surgery, the affected lung or lobe is removed at operation (pneumonectomy or lobectomy). Unfortunately the great majority of lung cancers are already inoperable when first diagnosed. In these cases *deep X-ray treatment* may sometimes give temporary relief.

In most cases no curative treatment is possible and all we can do is to relieve symptoms, to make the patient as comfortable as possible while he goes steadily down hill. Large doses of morphia or pethidine will be necessary if there is much pain from pressure on bones or nerves.

Prognosis.—The great majority of patients are dead within two years of diagnosis, whatever the treatment. A small proportion of those operated upon live in reasonable comfort for several years; a still smaller number are cured. In surgery lies the only hope of cure, and it is a small but increasing hope.

Case Histories

(1) Mr. G., aged 50, a grocer. He had had bronchitis for many years; he smoked 25 cigarettes a day. He attended the chest clinic for regular examination and X-ray. At one examination a dense shadow was found in the right lung, which had not been present in previous films; he had had no new symptoms. He was found to have inoperable carcinoma of the lung. He went downhill fairly rapidly about 3 months later and died 6 months after diagnosis.

(2) Mrs. A.A., aged 62. Had had cough, sputum, breathlessness and loss of weight for 3 months. She was a non-smoker. On examination at the chest clinic she was extremely ill and breathless. X-ray showed massive collapse of the right lung (Fig. 26). Bronchoscopy showed inoperable cancer of the right main bronchus. She died 3 weeks after her first X-ray.

A Note on Smoking

There is overwhelming evidence that cigarette smoking is the main cause of lung cancer. The disease is rare among non-smokers, common among heavy smokers, with the death rate among light smokers coming in between. A man who has always smoked heavily has about one chance in eight of dying of lung cancer; his non-smoking neighbour has a one in three hundred chance—nearly forty times less. Those who stop smoking improve their chances of escaping the disease. Pipe-smoking is less dangerous than cigarette smoking. Inhaling, curiously enough, seems to make no difference.

Cigarette smoking has increased enormously over the past few decades, and the staggering rise in lung cancer death rate appears to be the result. About twenty-five years' smoking is necessary to produce a cancer. Twenty-five years ago not many women were heavy smokers—this is thought to be the reason why women less often have lung cancer than men. The death rate among women is, however, rising.

We have seen that lung cancer is one of the least curable of malignant growths; yet it is the most preventable. The majority of the 19,119 who died of the disease in 1957—many of them men in their forties and fifties—would be alive today if they had never smoked.

Smoking is not only the main cause of lung cancer—it helps to cause a number of other diseases or at least make them worse—for

instance coronary artery disease, chronic bronchitis, obliterating arteritis and peptic ulcer.

Young people should know these facts before deciding to start smoking.

(7) Pneumoconiosis

The name means a lung disease caused by dust, such as silica dust (silicosis) or asbestos dust (asbestosis). Inhalation of such dust over a period of years may cause a progressive *fibrosis of the lung*.

Silicosis

This is the commonest variety of pneumoconiosis in this country. Silica is found in most forms of rock and stone. Miners, quarry men and metal grinders may develop silicosis after inhaling silica dust for years. The main symptom is *breathlessness*; the patient may also cough and wheeze. The diagnosis is made on *X-ray of the chest*, which shows a characteristic *fibrosis of the lung*.

The fibrous tissue extends and becomes denser over the years and the patient becomes increasingly breathless. He may finally die of respiratory or cardiac failure. *Tuberculosis* is a fairly common complication of silicosis.

Treatment.—No treatment will cure or even arrest the fibrosis of the lung in silicosis. *Prevention* is therefore all the more important. Dust accumulation can be reduced by good ventilation, moisture, etc. Workers in industries where there is a known risk of silicosis are usually X-rayed once a year. If the X-rays show any signs of silicosis the man is advised to change his job.

Should tuberculosis develop the usual treatment (p. 117) is given.

Workers disabled by silicosis can claim compensation. Each case is referred to an impartial tribunal—the Pneumoconiosis Panel—for assessment. Curiously enough, a man may be very breathless with little to show on X-ray, while another has few symptoms though his X-ray is grossly abnormal.

(8) Spontaneous Pneumothorax

Sudden *pain in the chest* followed by *rapidly increasing breathlessness*—these are the symptoms of spontaneous pneumothorax. The patient is usually a previously healthy man; the symptoms often come on after some sudden exertion or fit of coughing.

Investigations. *X-ray of chest* shows that there is a collection of *air in the pleural cavity*. The underlying lung is partially or completely collapsed—in severe cases it may be the size of a fist or smaller.

The heart may be pushed over towards the opposite side of the chest.

Explanation. A sudden tear in the surface of the lung allows air to escape into the pleural cavity and causes the pain in the chest. The

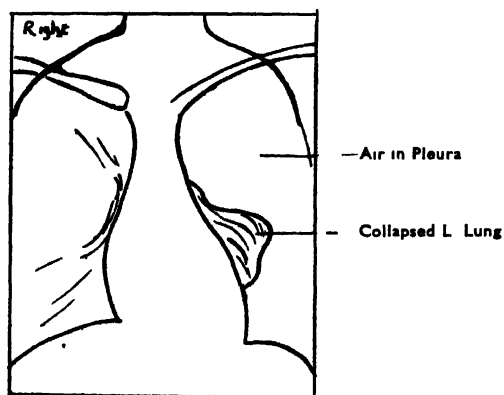
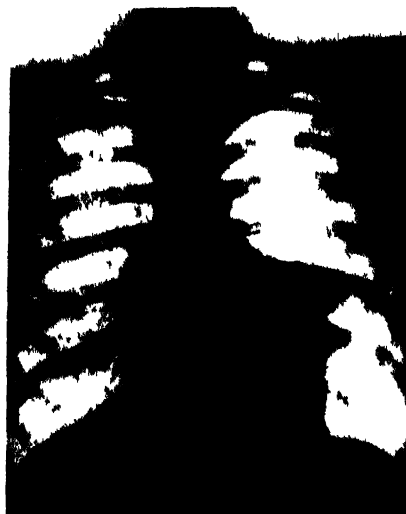


Fig 32 Spontaneous Pneumothorax
Left lung is completely collapsed and left pleural cavity is filled with air (Dr Luntz's case)

patient becomes increasingly breathless as the underlying lung collapses, and still more so if the heart and mediastinum* are pushed over and affect the function of the other lung.

*The structures between the two lungs.

The usual site of the tear is an *emphysematous bulla*—a bubble-like pocket of air on the surface of the lung. Very often the rest of the lung is normal. Spontaneous pneumothorax may also occur in generalized emphysema, pulmonary tuberculosis, lung abscess and other lung diseases.

Further Course

Mild Cases.—Symptoms clear up rapidly as the air in the pleural cavity absorbs, and the lung re-expands; presumably the torn lung heals.

Severe Cases.—The pneumothorax persists because air continues to escape from the torn lung; the patient may become severely breathless and distressed. The torn lung may bleed, so that blood collects in the pleural cavity as well as air. Occasionally, air may escape into the subcutaneous tissues (subcutaneous emphysema). This can be recognised by the unmistakable crackling sensation which it gives to the examining hand. Should the air penetrate into the mediastinum and track up into the tissues surrounding the larynx the danger of suffocation is acute.

Treatment. In mild cases a few days rest in bed is all that is required. In severe cases, where the air is slow to re-absorb, it may have to be removed. (a) A special needle with a trocar is inserted into the chest, the trocar removed and the air aspirated with a syringe. The needle is then withdrawn. (b) Another method is to connect the needle with an underwater drain; air bubbles out whenever the pressure inside the pleural cavity rises, and the air is gradually expelled. (c) If this fails, continuous suction for a few days may be tried. (d) An irritant solution such as silver nitrate is sometimes injected into the pleural cavity so that adhesions may develop between the two layers of pleura when the lung re-expands; this helps to prevent further pneumothoraces.

If hæmorrhage should occur the blood is aspirated; blood transfusion may be necessary, followed by operation.

If the patient has repeated pneumothoraces *thoracoscopy* may be necessary. A thoracoscope is an instrument through which the pleural cavity can be viewed; it is inserted through a small incision in the chest wall. The source of the air leak can often be discovered in this way. Surgical treatment may be necessary to remove bullæ or to mend tears which have not healed spontaneously.

After the lung has re-expanded the patient may require further investigation to exclude tuberculosis.

Case History. A.E. Aged 23. Five days before admission he was awakened in the night by pain in the left chest. He gradually became increasingly breathless; he went to work next morning but had to be sent home. X-ray showed spontaneous pneumothorax with complete collapse of left lung (see Fig. 32). Air was withdrawn by means of an intercostal catheter. The lung re-expanded completely within a fortnight. Follow-up X-rays were all normal.

(9) Pulmonary Embolism

Pulmonary embolism, or the blocking of a lung artery, has been mentioned several times as a fairly common and very serious complication of various diseases. The block is usually caused by a piece of blood clot which forms in the right side of the failing heart or in the leg veins (see p. 92), and later breaks off and is swept into the circulation.

If a large pulmonary artery is blocked the patient suddenly becomes intensely blue and breathless with acute pain in the chest; he may rapidly become unconscious and die in a few minutes. When only a small artery is affected the patient complains of a slight pain or "stitch" in the chest, and he soon develops an irritating cough with, perhaps, blood-streaked sputum. All degrees of severity are seen between these two extremes.

In severe cases the *E.C.G.* may help in the diagnosis, as the tracings show characteristic changes.

Treatment.—Oxygen for cyanosis, morphia for restlessness and pain, and venesection for venous engorgement may be required in the severe cases. Heart failure, which may either cause or follow pulmonary embolism, must be treated along general lines. *Anti-coagulants* may be used in certain cases—e.g. dicoumarol or tromexan (p. 76). The formation of further clots may thus be prevented, and it is said that the thrombi already formed clear up more quickly.

The *prevention* of pulmonary embolism is that of venous thrombosis (p. 93).

A Note on Haemoptysis

Haemoptysis, or blood-spitting, is a symptom which always requires investigating. When frank bleeding into the lung occurs the blood often wells up into the throat and the patient describes how he suddenly found his mouth was full of blood. Blood-streaked sputum, brought up after a violent fit of coughing, is less likely to indicate serious lung disease; it is fairly common in bronchitis and other chest infections. Blood in the sputum may come from the throat, or from bleeding gums, or from a nose bleed.

Almost any *inflammatory* or *malignant disease* of the lung may cause hæmoptysis, because lung blood vessels may be weakened and eroded in these diseases. Thus, hæmoptysis is common in pulmonary tuberculosis, bronchiectasis, lung abscess, some forms of pneumonia, and lung cancer. Hæmoptysis also occurs in *circulatory disorders* causing congestion of the lungs, such as mitral stenosis, and in pulmonary embolism. *Blood diseases* can cause hæmorrhages from the lung, as from other sites.

In the writer's experience, a brisk hæmoptysis is commonly the result of tuberculosis, lung cancer, bronchiectasis or pulmonary embolism. Small hæmoptyses are quite common in bronchitis. In a surprisingly large number of cases no cause can be found.

(10) Bronchitis and Emphysema

Acute Bronchitis

Acute bronchitis is common at all ages during the winter months, but it is more frequent and more severe among the young, the old and the weakly. It often occurs in the course of another disease; it may follow a bad cold, it is practically invariable in measles, whooping cough and typhoid, and it is common in nephritis and in influenza and many other fevers.

The onset of acute bronchitis occurring as an independent disease is generally rapid, with cough and a few degrees of fever. The cough causes a rasping sensation behind the sternum and the whole chest may feel sore. There is no sputum at first and the useless cough is wearing and distressing. Later on the patient begins to bring up sputum which is scanty and sticky at first but which soon becomes more copious. It is yellowish or greenish white in colour. The temperature returns to normal within a few days, and after a week or so in bed the patient rapidly makes a complete recovery in an uncomplicated case. In severe cases the symptoms may become worse owing to the development of pneumonia, when the outlook become much more serious. *Rapid breathing* is the most important sign of this development.

The symptoms of bronchitis are due to inflammation of the *bronchi*, the air passages leading from the trachea (windpipe) to the lung tissue. The inflammation is often caused by a mixed lot of organisms. The irritation caused by the congestion and inflammation of the mucous membrane lining these passages causes the cough, which is dry and painful at first. Later on the inflamed mucous membrane oozes with pus and excess mucus which are coughed

up as sputum. In all cases of bronchitis, acute or chronic, the bacteria, originally limited to the bronchi, may eventually infect surrounding patches of lung tissue, causing bronchopneumonia.

The *treatment* of acute bronchitis is that of any other feverish illness—rest in bed, good nursing, a light diet and plenty of fluids to drink. In the early stages inhalations of steam flavoured with menthol or Friar's balsam help to loosen sticky mucus. Inhalations can be given by means of an inhaler or the homely jug and towel. A *steamkettle* is useful in treating a child with bronchitis.

Drugs used in bronchitis.—A course of penicillin or sulphonamides is usually given and may shorten the illness, though their effect is much less dramatic than in pneumonia. Many other drugs are used in the treatment of bronchitis, either with the intention of promoting the flow of sputum or of stopping a useless cough. The former are called *expectorants*, the latter include the *linctuses*.

A patient with a dry cough or who is bringing up tough, sticky sputum longs for something to "cut the phlegm" or "loosen the cough," and many drugs have been given with this end in view. Those few "expectorants" which have been thoroughly tested appear to have no effect whatever on the volume of sputum produced. However, they are still prescribed in the common expectorant mixtures, and sometimes appear to give temporary relief. Iodides, ammonium salts and ipecacuanha are some of the common expectorants.

A *linctus* is a medicine which tends to stop coughing. If there is sputum to bring up it is wrong to stop the cough except when it is disturbing sleep because, in the absence of coughing, septic material would accumulate in the chest. If there is no sputum or if the patient is exhausted by coughing a linctus or some other cough reliever is indicated. Experience shows that any sweetish liquid temporarily allays coughing; linctuses are, therefore, nearly always sweet to taste. They also contain various other ingredients besides syrup, but the only drugs which really stop a cough are morphine and drugs whose action is similar—for example, codeine and amidone (physeptone). Gee's linctus, *linctus camphoræ* co. and *linctus scillæ opiatæ* all contain small doses of morphine. Linctus codein and amidone are excellent; heroin, though potent, is too habit-forming for safety. Another useful cough reliever is Dover's powder, of which 10 gr. contains $\frac{1}{16}$ gr. morphine.

Bronchitis in children

Acute bronchitis is common in young children, especially in the

course of the common fevers. Weakly children often suffer from recurrent attacks of bronchitis. Such repeated attacks are dangerous and may lead on to bronchiectasis. Drugs are of little use in treating these children. Good food and a healthy life—as far as possible in the open air—are what they need. Breathing exercises are often very helpful.

A period at an Open Air School, away from the constant risk of infection in an overcrowded home, may be of great benefit.

Chronic Bronchitis and Emphysema

These disabling diseases are extremely common in our country and cause an enormous amount of illness and loss of working time. There are said to be about a million sufferers in Britain, of whom over 25,000 die every year—more than the death rate from lung cancer, and nearly five times the death rate from tuberculosis of the lungs. The bronchitis death rate in Britain is the highest in the world—it is, for instance, about twenty times that in Scandinavia. This is certainly due, in part at least, to the *heavily polluted air* over our industrial towns—bronchitis is much commoner in towns than in the country, and is always commonest in the smokiest districts. *Cigarette smoking* also helps to cause bronchitis—the disease is much commoner and more severe among heavy smokers than among non-smokers. Bronchitis is commoner among *working class men* than in the professional classes (the severe forms are rare among women).

Most bronchitis deaths occur during the winter—cold and damp make the disease worse. But *fog* is worst of all. Bronchitics deteriorate, and many die, after a spell of foggy weather. The great fog which hung over London from December 5th–8th, 1952, caused approximately 4,000 deaths—more than any epidemic since the great cholera outbreaks of a century ago.

Chronic bronchitis usually starts insidiously as a mild winter cough with sputum in a middle-aged man. At first the sputum is scanty and consists largely of mucus; later it becomes more *copious and purulent*. Acute exacerbations are common, during which much purulent sputum is brought up. The bronchitic is often *wheezy* and finally becomes increasingly *breathless* and *cyanosed*. Symptoms are always worse during the winter—especially in foggy weather—but, as time goes on, cough and breathlessness persist throughout the year. Finally, the symptoms often merge imperceptibly with those of congestive heart failure.

In chronic bronchitis the walls of the bronchi are irritated by smoke, dust, “smog” and tobacco smoke. As a result an excess of mucus

is poured out. This mucus is coughed up as *sputum*, which is clear at first; later, the mucus becomes infected by various organisms and the sputum becomes *purulent*. The irritation of the bronchi causes *spasm of the bronchial walls*, as in asthma—hence the wheezing. In severe cases *emphysema* is nearly always present to aggravate the condition.

In emphysema the alveoli (air sacs) of the lung are distended with air and many of their walls break down. Many little blood vessels are obliterated when the alveolar walls rupture, and this impairs the oxygenation of the blood. Also, the obliteration of lung capillaries increases the load on the right ventricle, which has to pump blood round a partially obstructed circuit. After years of this, right heart failure supervenes, with the usual cyanosis, venous engorgement and oedema. (See *cor pulmonale*, p. 77).

The emphysematous patient can be recognised by his barrel-shaped chest, caused by the displacement of the ribs and sternum by the over-inflated lungs. The chest eventually becomes fixed in this position, and the patient has so little freedom of respiratory movement, his lungs are so inelastic that he easily becomes short of breath. And, because the blood gets too little oxygen, the emphysematous patient *is usually cyanosed even if the heart is not failing*.

Diagnosis of Chronic Bronchitis.—The history and clinical signs usually make this clear. But, as a precaution, every bronchitic should have his chest X-rayed in case he also has tuberculosis. Many a grandfather's cough has been labelled bronchitic until a grandchild living in the house has developed tuberculous meningitis.

Treatment.—Bronchitics should *give up smoking* and should avoid exposure to *fog*, and *damp*, and *extreme cold*. These measures are far more important than medicines and pills—but, unfortunately they are very much harder to apply. The bronchitic should have a job where his working day is spent at an even temperature. In very cold weather he should have a bed downstairs or in a heated bedroom. (The freezing British bedroom is harmful in this condition—the sudden change from the warm sitting-room often causes uncontrollable paroxysms of coughing.)

Bronchitics who are overweight should go on a reducing diet. Breathless people cannot afford to carry useless weight and, as we shall see (p. 250), fat in the abdomen pushes up the diaphragm and further reduces the possible lung expansion.

Drug Treatment.—(a) *Antispasmodics*—ephedrine, aminophylline and isoprenaline all act as broncho-dilators and help to relieve wheezing. Isoprenaline can be given by mouth or inhaled from an

atomiser; inhalations may also contain mucolytic (mucus-dissolving) hormones which are said to liquefy the sputum. Ephedrine is given by mouth, and, as we have seen, aminophylline can be given in tablet form or as a suppository if tablets make the patient sick.

(b) *Chemotherapy*.—Acute exacerbations with copious, purulent sputum, usually respond to antibiotics—for instance, penicillin and streptomycin, or tetracycline. Prolonged courses of antibiotics are occasionally recommended. If a patient has severe bronchitis with much purulent sputum, a small dose of tetracycline (0.25 g. twice daily throughout the winter) may enable him to keep going when otherwise he would be off work for weeks at a time. Cases for prolonged chemotherapy need careful selection and regular medical supervision.

(c) *Symptomatic Treatment*.—The average bronchitic would feel lost without his “bottle of medicine” (expectorant or linctus). Actually this is the least important part of the treatment, though it is often comforting.

Treatment of Heart Failure in Chronic Bronchitis.—This has already been described (p. 77). In brief, the patient is given oxygen, with caution; respiratory stimulants if necessary; antispasmodics and diuretics as required; and, for the chest infection, full doses of antibiotics. Morphia is forbidden and all other sedatives cut down to a minimum.

Prognosis.—A winter cough at 40, long periods off work in the fifties and disablement at 60 are the usual story. The bronchitic rarely lives to three score years and ten, and heart failure or an acute respiratory infection may be fatal much earlier. But the outlook is very much brighter if, in the early stages he can reduce the irritation to his bronchi as outlined above.

The problem of clean air is a national one. Just as pollution of the drinking water once caused thousands of deaths from cholera and typhoid, so air pollution now causes thousands of deaths from bronchitis. Clean air legislation has made a start, but we have a long way to go before bronchitis is wiped out as was cholera nearly a hundred years ago.

Case History.—Mr. T., a labourer, aged 56, first complained of a troublesome winter cough in 1935. He carried on until March, 1941, when an acute attack kept him off work for a month. In February, 1946, he suddenly became much worse, with severe cough, breathlessness and swelling of the ankles. He had signs of severe bronchitis and of congestive heart failure, deep cyanosis, œdema, venous congestion and a rapid,

feeble pulse. After a month in hospital for treatment of the heart failure he was still only fit for a semi-invalid life. Two months after discharge he was going out for short walks but was quite unfit for work. He returned to work for a few months in 1947; in 1951 he died after two or three years' semi-invalidism.

(11) Asthma

Asthma is a common condition in which, for many years, a person is subject to sudden attacks of difficulty in breathing and wheezing. Asthma often runs in families; asthmatics may suffer from urticaria, eczema or hay fever, or these conditions may affect other members of the same family. It is commoner among men than among women, and seems to attack principally the intelligent and the over-anxious. It is said (proudly, by asthmatics) that *no fool has asthma*.

An asthmatic patient may be admitted to hospital for investigation, for the treatment of complications or, as an emergency, during an unusually severe and prolonged attack.

An asthmatic attack may come on at any time, but most frequently occurs at night. The patient suddenly awakes with a sense of suffocation and acute difficulty in breathing. He starts up in bed; each breath is drawn in quickly and forced out with great difficulty to the accompaniment of loud wheezing noises. "Well, if I can get this breath once out, I'll take care it shall never get in again!" an asthmatic friend exclaimed to Jonathan Swift. The chest is distended and the muscles of neck and shoulders stand out with the strain. The patient looks pale and anxious, with bluish lips and distended neck veins. Sweating may be profuse. Towards the end of the attack there may be a bout of coughing when the patient coughs up little whitish pellets of tough mucus rather like boiled sago grains. The attack may last for a few minutes to several hours, after which the patient often sleeps till morning. Sometimes one attack follows another without a break for many hours or even days—*status asthmaticus*.

What is it that causes the asthmatic thus to start up out of a peaceful sleep, desperately fighting for breath? The difficulty in breathing is caused by a sudden narrowing of the bronchi, due to spasm of the muscle in the bronchial walls. The spasm is due to overaction of the nerves supplying the bronchial walls—the *vagus nerves*. What, then, accounts for this vagal overaction which appears to be the cause of all the trouble?

Here we are on much more uncertain ground. It appears that asthmatics inherit an unduly sensitive set of bronchi, and that stimuli which have no effect on normal people send this irritable

bronchial system into spasm. All sorts of stimuli may trigger off this spasm. Some asthmatics are sensitive to cat-fur or horse-hair, or feathers, others to certain pollens; another group reacts to food, such as eggs, shellfish or chocolate. When very small quantities of a substance like one of these provoke an attack the person is said to be *allergic* to it. Allergy is not confined to asthma; in other conditions such as hay-fever, eczema or urticaria, attacks may be precipitated by contact with substances which are harmless to normal people.

In addition to allergy, *respiratory infection* (e.g. sinusitis, bronchitis) may be associated with asthma. Finally, many attacks are due to psychological causes—worry, outbursts of temper or the mere expectation of an attack, as in the famous case of the man who was sensitive to roses and who had an asthmatic attack on being handed an artificial rose.

In between attacks the young asthmatic usually seems perfectly healthy though he may wheeze from time to time. In later life asthmatics generally become increasingly breathless on exertion, owing to the development of complications such as bronchitis and emphysema, and, finally, cor pulmonale.

Treatment.—We do not know how to change the asthmatic diathesis (constitution), which appears to be inborn. If a child's asthmatic attacks can be prevented for two years or more he may never have another, but adults are seldom so fortunate and are usually subject to attacks for life. However, their frequency and severity can be reduced by treatment, and every asthmatic should be enabled thus to live an active life. First of all he should avoid anything which he has found by experience to precipitate an attack, whether it be cats, hayfields or oysters. If a patient is sensitive to horse-hair or feathers he should use flock or kapok pillows and mattress and dispense with an eiderdown.

A series of *sensitivity skin tests* is sometimes helpful. For these we need a set of solutions, of feathers, cat fur, house dust, pollen, various foods, and so on. A drop of each solution is placed on the skin of the forearm and a scratch is made through the drop (or the solutions can be injected intradermally). If the patient is sensitive to any of these substances a red flare or wheal develops at the site of the scratch test or injection. A series of *desensitising injections* may then be given, starting with a minute dose of the offending substance and gradually increasing the dose.

In addition to avoiding substances to which he is allergic the asthmatic should be treated for any abnormality or infection of the nose or throat which is considered to affect his asthma. He should avoid

overeating last thing at night, overwork and worry. Anxious parents should be discouraged from fussing over their asthmatic children, who can easily be turned into self-pitying hypochondriacs. An open-air school or a long stay in the country is often advisable. *Breathing exercises* correct the deformities of chest and spine so common in asthmatics, and strengthen the muscles of respiration. Small doses of one of the bronchial dilators described below may be taken two or three times a day when attacks are recurrent. *Antihistamine drugs* (see p. 269) have been tried but are usually disappointing. Sedatives, such as phenobarbitone, may be given if the patient is over anxious and tense. Potassium iodide mixtures seem to be helpful when there is much cough.

Treatment of the Asthmatic Attack.—For these attacks *adrenaline*, *isoprenaline* or *ephedrine* are usually given as they counteract the vagus nerves and tend to make the bronchi relax. Ephedrine gr. $\frac{1}{2}$ – $1\frac{1}{2}$ may abort a mild attack. Isoprenaline sulphate can be administered in the form of tablets which are allowed to dissolve under the tongue. The dose is 10–30 mgm., or more in severe attacks. Isoprenaline can also be *inhaled* from an atomiser; this may rapidly end an attack. Another solution which may be used in this way contains atropine, adrenaline and other drugs. In severe attacks an injection of adrenaline is generally given (3–8 minims, repeated if necessary after 5–10 minutes.)

Status Asthmaticus.—In severe cases the patient is nursed in an oxygen tent. An intravenous injection of *aminophylline* may bring the attack to an end. *Steroids* often have a dramatic effect, and may be life-saving. Cortisone and prednisone are given by mouth; an intravenous or intramuscular injection of hydrocortisone hemisuccinate may be given in very severe cases.

Should a sedative be necessary in status asthmaticus paraldehyde in small doses given by intramuscular injection is safe and effective.

Hundreds of asthma cures have been recommended. One and all they result in 80 per cent. of cases cured—for the time being. Such is the effect of faith in a condition so much influenced by the mind.

Sir Arthur Hurst, a physician who was a life long asthmatic, described how, with his wife, he visited a famous spa one year with excellent results. Next year he went alone, was very unhappy and became much worse. On rejoining his wife in Paris his attacks cleared up completely, although he ignored every regulation that had been laid down for his observance.

The outlook for the asthmatic.—As already mentioned, except in childhood, asthma is unlikely ever completely to leave one born

subject to it. Elderly asthmatics generally develop bronchitis and emphysema, which may cause right heart failure, as described in the previous chapter. Asthma definitely shortens life. However, with good sense and good management the asthmatic should be able to live an active life for many years, and doctors and nurses should see to it that he knows "how to be happy in spite of the bad luck of having been born with the asthma diathesis." (Hurst.)

Case Histories.—Private N., A.T.S., had her first asthmatic attack while attending a drivers' training course soon after enlistment. Tests showed that she was above average in intelligence, and she was most anxious to become a driver and to do well in the A.T.S.—not at all a neurotic type. She had to be down-graded, however, to a medical category requiring a change of army occupation. She was most sensible about her asthma, and would take her ephedrine when she felt an attack coming on and sit quietly in Sick Bay till it was over. There was no family history in this case, and we were unable to find any precipitating cause of her attacks.

(2) J.G., aged 6, suffered from asthma from the age of two. A year later he was given a kitten; he then wheezed almost continuously for a fortnight. The kitten was given away and he immediately improved. He continued to have attacks if he was in a house with a cat, and also if he had a cold or sore throat.

(3) P.L., aged 8, had eczema as a baby. At the age of 18 months he had a very severe attack of pneumonia. He has had frequent attacks of asthma ever since. He still has flexural eczema.

DISEASES OF THE BLOOD, SPLEEN AND LYMPH GLANDS

Anæmia

IN every ward—medical, surgical, pædiatric or gynæcological—nurses are likely to have anæmic patients under their care. Anæmia, or lack of blood, is very common. A severe anæmia may be the main reason for the patient's admission to hospital; or anæmia may complicate some other illness—such as rheumatoid arthritis or nephritis. Many thousands of mildly anæmic patients are treated as out-patients, or by their private doctors.

The *symptoms* of anæmia are much the same, whatever the cause—and anæmia has many causes, as will be seen. The first symptom is often *shortness of breath*. *Tiredness, irritability, and difficulty in concentrating* are all common, the patient may at times feel giddy or faint, and his ankles may swell. In severe cases he may be confused, he is very susceptible to infection and, as we have seen, he may suffer for anginal pain or frank heart failure. Patients who gradually become anæmic over a period of years do not always complain of any of these symptoms, and may be surprised when a routine blood count discovers a fairly severe anæmia. If the anæmia is then treated, however, they are often delighted and surprised by their renewed vigour.

The severely anæmic patient is very pale, but pallor is not always noticeable in milder cases of anæmia, and some people are always pale, though not anæmic. The colour of the conjunctiva inside the lower lid is a better guide than the colour of the face, and so is the colour of the creases in the palm of the hand. (Stretch the skin of the palm by bending the fingers back, and compare the colour of the patient's creases with those of your own palm.) The anæmic patient usually has a rapid pulse rate. In very severe anæmia he is likely to be *feverish*.

Blood tests are always necessary if anæmia is suspected. The simplest test is a *hæmoglobin estimation*; often this is done first, and, if abnormal, a full blood count follows. Normal blood contains about 15 G. hæmoglobin per 100 ml.—rather less in women than in men. (14·8 G. per 100 ml. is often referred to as Hb. 100 per cent). In anaemia the blood contains less hæmoglobin than this—often very much less.

Explanation of the symptoms.—These are all the result of the lack of hæmoglobin. Hæmoglobin is the pigment which gives the blood its colour. It is the oxygen carrier of the blood—in the lungs it combines with oxygen which is then transported to every part of the body as the blood circulates. In severe anæmia the blood cannot carry enough oxygen to supply the needs of the tissues. The *heart* is particularly sensitive to oxygen lack—hence the breathlessness and swollen ankles of the anæmic patient, and, in occasional severe cases, the anginal pain. Irritability, loss of concentration and faintness result from the poor oxygen supply to the brain. Tiredness may have the same cause, or may be the result of oxygen lack in muscles and other tissues.

We come now to an account of the more important types of anæmia. A short classification of the anæmias will be found at the end of the chapter.

(1) Anæmia Due to Loss of Blood

A person may become anæmic as a result of slow, continued bleeding (chronic hæmorrhagic anæmia) or a sudden severe hæmorrhage.

Chronic Hæmorrhagic Anæmia.—Common causes are bleeding hæmorrhoids (piles), heavy periods and repeated small hæmorrhages from a peptic ulcer. A growth of the stomach or bowel may also bleed and cause anæmia. *Aspirin* can cause gastric bleeding, and it has been shown that people who take aspirin regularly may become anæmic because of this.

It must be remembered that the patient may be unaware of these bleedings, or at least of their extent. Small gastro-intestinal hæmorrhages are particularly likely to go unnoticed.

Symptoms are those of anæmia in general. The more gradual the onset of anæmia the less the patient is likely to complain of any symptoms.

Investigations. Blood Count.—The hæmoglobin may be very low (below 7 g. per 100 ml.). The *red cells*, which carry the hæmoglobin, are reduced in number, but not to the same extent. This means that the red cells contain less hæmoglobin than usual and look pale in stained films. The cells are also apt to be small. This type of anæmia is therefore described as *hypochromic** and *microcytic*†.

Here is a typical blood count in such a case (omitting the white cell count); the normal values are shown for comparison.

*Hypo = below; chrome = colour. †Micro = small; cyte = cell.

	<i>Normal</i>	<i>Hypochromic Anæmia</i>
Hæmoglobin (Hb)	14·8 G. = 100% per 100 ml. blood	7·4 G. = 50%
Red cells (R.B.C.)	5 million per c.mm. blood	4 million per c.mm.
Hæmatocrit (Volume of packed red cells per 100 ml. blood)	42	30
Colour Index (Calculated from the Hb. and R.B.C.)	1	0·6
Mean corpuscular hæmoglobin concentration (M.C.H.C.) calculated from the Hb. and hæmatocrit	34	25
Mean corpuscular volume (M.C.V.) calculated from the hæmatocrit and R.B.C. count	87 cubic μ	75 cubic μ

The low colour index and M.C.H.C. show that the red cells are *too pale*; the low M.C.V. that they are *small*.

Explanation.—When blood is lost slowly over a long period, fluid is drawn into the blood stream from the tissue spaces, so that the blood volume remains constant. Hence the blood becomes more and more dilute, and will contain fewer red cells and less hæmoglobin than normal blood. Now whenever the blood is carrying less oxygen than normally, the blood-forming tissues in the bone marrow speed up the production of red cells, and this helps to make good the deficiency. But, after a hæmorrhage, it is easier to produce new red cells than to fill them with hæmoglobin, because hæmoglobin contains iron, of which the body has no reserve. Hence, in chronic hæmorrhagic anæmia, the cells are pale (hypochromic) and the hæmoglobin is reduced more severely than the number of red cells.

Further investigations (such as a barium meal) may be necessary to discover the source of the bleeding.

Treatment

(a) The source of the bleeding must be found and treated, either medically or surgically. If the patient is anæmic because repeated doses of aspirin have caused gastric bleeding, the aspirin must be discontinued, and another pain-relieving drug, such as phenacetin, substituted.

(b) *Treatment of the Anæmia.* If mild, or moderately severe, the anæmia will probably respond to treatment with iron; this will enable the bone marrow to produce more hæmoglobin and to send out enough well filled red cells to correct the anæmia *once the bleeding has stopped*. Iron may be given as ferrous sulphate (gr. 3), ferrous gluconate 5–10 gr.) or ferrous succinate (2½ gr.) thrice daily.

If the anæmia is very severe, blood transfusion may be necessary. This is always given *slowly*. This is because the patient has a *normal blood volume* and a *heart weakened by chronic lack of oxygen*. Suppose we suddenly increased the blood volume by two or three pints, this extra load on the weakened heart could precipitate heart failure and pulmonary œdema. So watch carefully the rate of flow of the blood transfusion in such a case. The drip is usually set at such a rate that a pint takes several hours to run in. Do not let it run faster—a sudden increase in the rate of flow could kill the patient.

Whenever possible *packed red cells* are transfused instead of whole blood, so that the blood volume is increased as little as possible.

Treatment with iron will probably be necessary after the transfusion.

Acute Hæmorrhagic Anæmia

This is caused by a *sudden loss of a large quantity of blood*. The cause may be a hæmatemesis or melæna, a violent nose bleed, a massive uterine hæmorrhage, rapidly bleeding piles, or, of course, an injury. Severe bleeding from the tonsillar bed following tonsillectomy can occur, and nurses in “T’s and A’s” wards must always be on the lookout for such bleeding, which may be fatal if not checked in time.

Symptoms of Severe Hæmorrhage.—The symptoms of anæmia are overshadowed by those of *shock*. The patient is pale, cold, sweaty, restless and anxious; he is breathless—so breathless that he may gasp for air (*air hunger*); he feels faint and may actually lose consciousness. The *pulse* is rapid and feeble and the *blood pressure* low. The *blood*

count gives no indication of the extent of the blood loss, and may be nearly normal. Estimations of the *blood volume* show that this is low.

These symptoms are mainly due to the sudden loss of a large volume of blood, for if the blood volume is suddenly reduced the circulation collapses; the heart is poorly filled with blood from the collapsed veins, the poorly filled heart beats feebly and a vicious circle is set up.

Treatment is a matter of urgency. *Blood transfusion* must be given *as soon as possible* and the blood run in *as fast as possible*. This is because we must restore the blood volume to normal in the shortest possible time if shock is to be relieved; several pints can often be given in half an hour.

(Note the contrast to transfusion in chronic hæmorrhagic anæmia—the rule is always: slow bleeding, slow transfusion; rapid bleeding, rapid transfusion.)

Measures to stop the bleeding may be necessary.

Meanwhile, the patient is nursed *lying flat* or with one pillow; in severe cases the foot of the bed is raised on blocks to improve the blood supply to the brain. *Oxygen* may be necessary in severe cases. A *sedative*—morphia or soluble phenobarbitone—may be given to relieve distress and restlessness. *Do not try to warm the patient*—although his skin feels cold to the touch he is not aware of feeling cold. The skin is cold because its blood has been withdrawn to more vital structures. Hot water bottles, blankets and heat cradles reverse this process; the skin blood vessels dilate, the blood pressure falls still further, blood is withdrawn from vital organs and the symptoms of shock increase.

Before, during and after the blood transfusion and other active forms of treatment the nurse must keep a constant watch on the patient, observing, and reporting to the doctor, symptoms such as increasing pallor, restlessness or air hunger. A half-hourly or hourly pulse chart must be kept; frequent blood pressure readings may also be necessary. A rising pulse rate or falling blood pressure usually means continued bleeding.

The results of treatment are usually excellent if the bleeding can be stopped.

(2) Iron Deficiency Anæmia

This is probably the commonest type of anæmia in this country. The patient is most often the middle-aged mother of a working class family. On enquiry, she has the usual symptoms of anæmia, though

she may not complain of them spontaneously, for she is used to feeling tired and breathless. Asked about her diet, she admits that she does not like greens, cannot afford eggs, and gives her husband the lion's share of the Sunday joint.



Fig 33 Spoon shaped nails in iron-deficiency anemia

On examination, in addition to the usual signs of anæmia we may find that her *nails* are brittle and flattened or concave (*spoon shaped nails*) and that her *tongue* is red and sore.

The *blood count* shows that she has a hypochromic microcytic anæmia similar to that of chronic hæmorrhagic anæmia.

Gastric Analysis.—If carried out, this investigation often shows that the patient's gastric juice *lacks the hydrochloric acid* present in normal juice.

Explanation.—This patient's anæmia is due to a *shortage of iron*, of which not enough is absorbed for the production of normal quantities of hæmoglobin. First of all, her diet includes *too little iron containing food*. Then, the small amount of iron in her diet is *poorly absorbed*, because her gastric juice lacks hydrochloric acid, which normally helps in the absorption of iron. So too little iron is absorbed to make good the loss during menstruation, and for pregnancy and lactation. (Many of these women have particularly heavy periods.) As we have seen, iron is necessary for hæmoglobin production, and in iron deficiency anæmia the hæmoglobin content of the blood is more severely reduced than the red cells, resulting in hypochromic microcytic anæmia.

Treatment.—Iron deficiency anæmia usually responds very well to courses of iron by mouth—e.g. ferrous sulphate, gluconate or succinate. Treatment may have to be very prolonged—sometimes the patient is well advised to take iron on and off for life, as absorption of iron in food may be so poor.

The patient's diet should always be discussed, and she should be advised to eat plenty of the iron-containing foods.

Iron Deficiency Anæmia in Childhood

Children may also be anæmic because they eat too little of the iron-containing foods. This may be due to poverty or faddiness. The writer has seen a severely anæmic child of twelve in a good middle class home; although—or perhaps because—the mother fussed constantly about her children's health, the child was taking an almost iron-free diet as a result of her numerous fads and dislikes. Her hæmoglobin rose to normal after a few weeks of iron tablets, but her faddiness remained a problem.

Infants become severely anæmic if they are kept on an all-milk diet much after the age of six months. This is because the reserve of iron with which the baby is born is used up during the first few months of life. After this he needs iron-containing food (e.g. sieved greens, meat gravy); milk—especially cows' milk—contains very little iron.

Prevention of Iron Deficiency Anæmia

A good diet, as already explained, prevents iron deficiency anæmia in normal healthy people. Unfortunately, many iron-

containing foods are either expensive (eggs, meat, liver) or not very popular (greens). The average British diet contains enough iron for men but barely enough for women, whose iron supplies are subject to the additional demands of menstruation and child bearing; because of this, iron deficiency anæmia is almost exclusively a disease of women. If a woman's diet is just a little below average, because of poverty, food fads or wifely self-sacrifice, or if the need for iron is just a little raised, by pregnancy or heavy periods, she becomes anæmic. Nurses should urge their women patients to eat plenty of iron-containing foods, and to see that plenty is available for their children. Many housewives are very "vitamin conscious", though vitamin deficiency is extremely rare in Britain; iron deficiency is extremely common, but few housewives consider the iron content of the food they choose for themselves and their families.

(3) Pernicious Anæmia

Pernicious anæmia is so called because, before 1926, it was invariably fatal. It is not rare among elderly and middle-aged people; men and women, rich and poor, are equally affected. The patient complains of the usual symptoms of anæmia, which may come on so insidiously that he is severely anæmic by the time he seeks medical advice. He usually looks slightly yellowish as well as pale. He may also suffer from *indigestion* and a *sore tongue*, and also *tingling* and *pins and needles of the toes and fingers*. His *diet*, on enquiry, seems quite satisfactory.

Investigations.—Here is a typical blood count for such a man:

Hæmoglobin (Hb)	7·6 G/100 ml. = 52% (low)
Red blood corpuscles (R.B.C.)	2,000,000/cu.m. (very low)
Hæmatocrit	22 (very low)
Mean corpuscular hæmoglobin concentration (M.C.H.C.)	34 (normal)
Colon Index (C.I.)	1·3 (high)
Mean corpuscular volume (M.C.V.)	110 (high)

This blood count, in which the red cells are reduced more than the hæmoglobin, and in which the M.C.V., M.C.H.C. and C.I. are high or normal, tells us that the red cells, though few, are *large* and *well filled with hæmoglobin*. It may therefore be described as a *macrocytic*,* *hyperchromic*† anæmia.

* Macro = large; cyte = cell. †Hyper = above; chrome = colour.

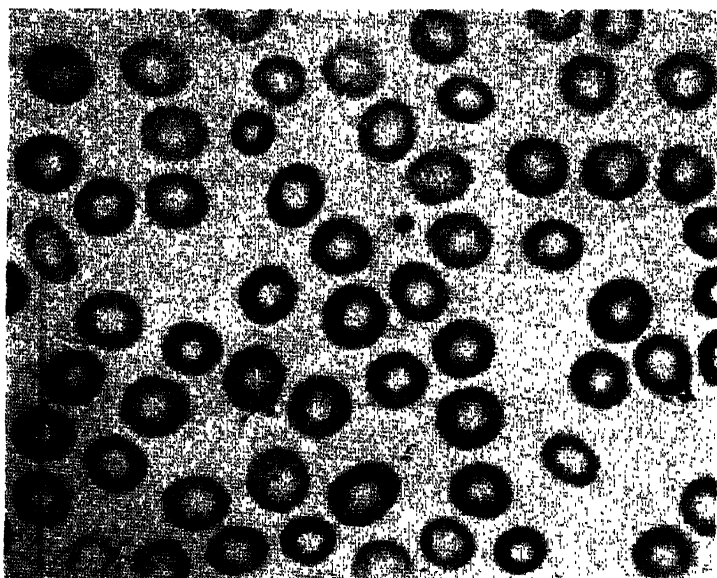


Fig. 33. Photograph of a blood film under the microscope, showing normal red blood cells.

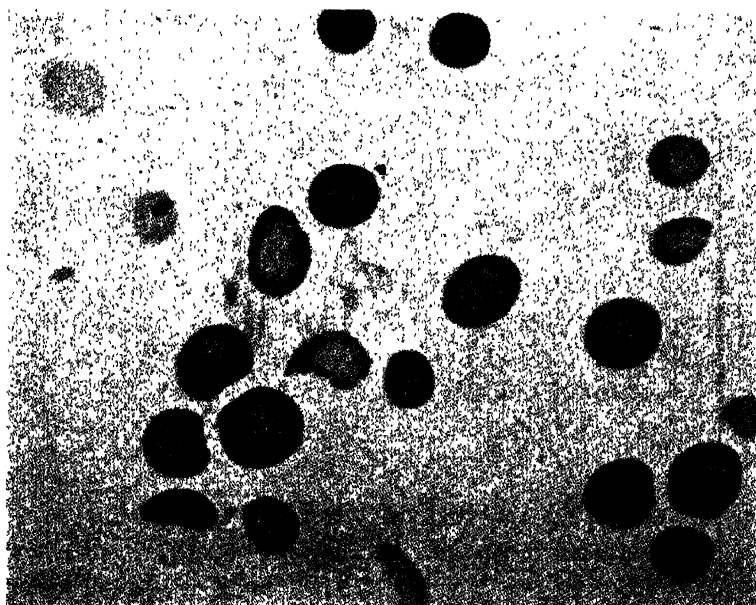


Fig. 34. Blood Film in Pernicious Anemia.
Note that many of the red cells are large, irregular and well filled with hemoglobin, as shown by their darker colour.

Gastric Analysis.—Gastric juice is aspirated after an injection of *histamine*, which provides the maximum stimulus to gastric secretion. In pernicious anæmia, not only hydrochloric acid but *all the digestive ferment* are missing.

Bone Marrow Biopsy.—Marrow can be withdrawn from the sternum or the iliac crest, using a special needle. The marrow is then stained and examined under the microscope. In pernicious anæmia the cells from which the red cells develop are large and abnormal.

Post Mortem.—Nowadays, deaths from untreated pernicious anæmia are extremely rare. Formerly they were common enough, and it was shown that deep red marrow filled most of the bones in the body, whereas in the normal adult red marrow is found only in a few bones, such as the sternum.

The Cause of Pernicious Anæmia.—This disease is due to *a disorder of red cell production*. Mature red cells can only be manufactured in the marrow in the presence of *vitamin B.12*, a substance present in minute quantities in many foods. In pernicious anæmia vitamin B.12 cannot be absorbed because the gastric juice is abnormal; as we have seen, it totally lacks hydrochloric acid and digestive ferment, and it also lacks the *intrinsic factor* which is necessary for the absorption of vitamin B.12. In the absence of vitamin B.12 the marrow, in its desperate efforts to keep up the supply of red cells, hurries into the circulation abnormal, immature, irregular cells, mostly of large size, and all crammed with hæmoglobin, of which there is no lack—hence the macrocytic hyperchromic anæmia.

The patient's *yellowish tinge* is explained as follows: red cells normally live for three or four months, after which they are destroyed. The hæmoglobin from these superannuated cells is broken down, and the yellow dye *bilirubin* is formed, which is got rid of by the liver in the bile. In pernicious anæmia the red cells are more readily broken down than are normal ones, and, as they contain so much hæmoglobin, an excess of bilirubin results, which gives the patient his slightly jaundiced complexion.

Nervous Complications in Pernicious Anæmia.—The pins and needles—in severe cases numbness and weakness of the legs—are due to a degeneration of the nervous tissue peculiar to this disease. This degeneration may affect the nerves—peripheral neuritis—and the *spinal cord*, when it is called *sub-acute combined degeneration of the cord*. A severe degree of *mental confusion* is sometimes seen. In advanced cases the patient may be completely paralysed, but happily such an event is rarely seen these days. The exact cause of this nervous degeneration is not known.

Treatment.—Before 1926 pernicious anæmia was invariably fatal. In that year Minot and Murphy, in the U.S.A., found that anæmia was relieved and life indefinitely prolonged by large doses of liver—half a pound, raw, daily was what the victims had to eat. We now know that liver relieves pernicious anæmia because vitamin B.₁₂ is stored in that organ. Later, liver extracts for intramuscular injection became available. Vitamin B.₁₂ was isolated in 1948, and is now always used in the treatment of pernicious anæmia. It is so potent that a millionth of a gramme (one microgramme, 1 μ g.) has a perceptible effect. The usual dose is 50–250 μ g. Large, frequent doses are given at first. Later the injections are given at weekly and then at fortnightly intervals. The patient will need injections every 2–4 weeks for the *rest of his life*. Regular blood counts are necessary to make sure that treatment is adequate.

The effect of vitamin B.₁₂ treatment in pernicious anæmia is dramatic. Within 48 hours the patient is feeling and looking better, and the blood count is improving. The blood is usually normal within about 6 weeks.

Nurses should impress upon their “P.A.” patients the absolute necessity of continuing with the treatment and blood counts—sometimes a patient feels so well that he thinks he is cured and no further treatment is necessary. Relapse is then inevitable, and nervous complications may develop. A properly treated “P.A.” patient should live his allotted span in normal health, with a normal blood count.

Other Macrocytic Anæmias

Anæmia, almost indistinguishable from pernicious anæmia, results from a shortage of *folic acid*, a substance present in liver, spinach, beet and many other foods. The main differences are that sore tongue is less common in folic acid deficiency, and nervous complications never occur; the gastric juice usually contains acid. The shortage of folic acid may be due to a *grossly inadequate diet* (as in certain tropical nutritional anæmias) or to *poor absorption*, as in *steatorrhœa*, after *gastrectomy* and other operations on the gastrointestinal tract, and in *macrocytic anæmia of pregnancy* (when a temporary disturbance of absorption may occur). The anæmia in all these cases usually responds to folic acid; in some cases it may respond to vitamin B.₁₂ too.

Idiopathic Steatorrhœa

This is the adult form of cœliac disease (p. 374); the patient may or may not have had symptoms of cœliac disease as a child. In the adult diarrhœa and fatty stools are much less troublesome than in

childhood, and the patient more often consults his doctor because of *anæmia*. The *anæmia* is macrocytic in type. Special investigations of the stools show that they contain an excess of fat.

Other food ingredients besides fat are poorly absorbed, notably folic acid—hence the *anæmia*. *Calcium absorption* may also suffer, causing soft bones (osteomalacia) or tetany (p. 236), and so may the absorption of vitamins. The patient is treated with folic acid, calcium and vitamins as required. A gluten-free diet may be as effective as in coeliac disease.

Steatorrhœa (fatty stools) may also occur in fibrocystic disease (p. 377), Crohn's disease (p. 181) and tuberculous enteritis (p. 182). A macrocytic *anæmia* may occur in severe cases of any of these diseases.

4. Aplastic Anæmia

This variety of *anæmia* is rare and deadly. *Drug reaction* is the commonest cause. Thiouracil, sulphonamides, chloramphenicol, gold salts, troxidone and tolbutamide have all been known to cause aplastic *anæmia*. Sometimes no cause can be found. In such cases the patient may eventually prove to be suffering from leukæmia (see below).

Symptoms.—The patient rapidly becomes severely *anæmic*, with fever, ulcerated throat and bleeding from the gums and other sites.

The blood count shows that not only the red cells are severely reduced, but also the *white cells* and the *platelets*—so that the symptoms of agranulocytosis (p. 154) and purpura (p. 151) are added to those of *anæmia*.

Marrow biopsy shows a complete failure of the blood-forming tissues, which have stopped manufacturing red cells, white cells and platelets.

Treatment.—If the patient is taking any drug which could cause aplastic *anæmia* this drug must, of course, be stopped.

Cortisone may be helpful, especially in the drug reaction cases. If *gold salts* were the cause *B.A.L.* (British anti-lewisite) acts as an antidote.

Repeated *blood transfusions* are life saving—or at least they prolong life.

Penicillin is given to control throat and other infections.

To control the bleeding resulting from the shortage of platelets, special *platelet transfusions* may be given—but such transfusions are difficult, and require a special apparatus.

Prognosis.—This is still grave, even with modern treatment.

(5) Hæmolytic Anæmia

In iron deficiency anæmia, pernicious anæmia and aplastic anæmia the marrow cannot manufacture enough red cells. In hæmolytic anæmia red cells are scarce because they are broken down too quickly (Haem = blood; lysis = breakdown).

Congenital hæmolytic anæmia is an inherited disorder which runs in certain families. Symptoms may come on at any age from infancy onwards. The patient suffers from an anæmia which fluctuates in severity: he is often a little jaundiced as well. Special blood tests show that the red cells are abnormally *fragile*, and so are broken up before their normal life span of three or four months is reached. As we have seen, the yellow pigment *bilirubin* results from the breakdown of hæmoglobin; when red cell breakdown is very rapid, enough bilirubin may be set free to cause mild jaundice.

Treatment.—Removal of the spleen (splenectomy) may relieve all symptoms, because many of the red cells are destroyed in the spleen.

Acquired hæmolytic anæmia. This may occur in malaria, incompatible blood transfusion, septicæmia, in toxic reaction to certain drugs and many other conditions.

Treatment is that of the primary condition. *Steroids* give striking relief in certain cases.

(6) Anæmia in Other Diseases

Severe anæmia is very common in rheumatoid arthritis, ulcerative colitis, bacterial endocarditis, puerperal sepsis and, indeed, in most



Fig. 35. The rash in thrombocytopenic purpura
From Fanconi and Wallgren's *Textbook of Paediatrics*, Heinemann

chronic infections. Many toxic conditions, such as chronic uræmia or lead poisoning can cause a severe anæmia, as can any malignant condition. It appears that most bacterial and some chemical poisons inhibit normal blood production or make the red cells more easily destroyed, or both. Such anæmia generally resists all treatment unless the primary disease is cleared up. In anæmia due to *infection*, as in ulcerative colitis, infected burns, etc., blood transfusions are often very valuable—not only do they cure the anæmia but they help the body to overcome the infection.

Anæmia also occurs in *scurvy* and *myxædema* owing to the lack of vitamin C and thyroid secretion respectively, both of which are necessary to normal blood formation. The anæmia in each case clears up as the primary disease is treated.

(7) The Bleeding Diseases

There is a large group of diseases in which the patient is apt to bleed spontaneously or after minor injuries. They bleed because the *blood clots poorly*, or because the *capillaries are faulty*, or both.

Purpura

In purpura the patient has a rash of *purple spots or blotches* (hence the name). These spots are caused by bleeding from the capillaries under the skin. Bleeding into other tissues may give rise to deep hæmatomata. The patient may also have attacks of hæmorrhage from the nose (epistaxis) or kidney (hæmaturia) or uterus (menorrhagia). *Melæna*—the passage of tarry stools—shows that blood has been shed in the stomach or upper intestine. Bright red blood in the stools means that the patient is bleeding from the rectum or colon.

Purpuric rashes occur in many fevers, for instance cerebrospinal meningitis (p. 324) and, rarely, in cases of measles, scarlet fever or smallpox, when the infection is of overwhelming severity. Purpura occurs in aplastic anæmia and leukæmia, and sometimes in patients dying of cancer or chronic nephritis. Certain drugs may cause purpura in sensitised patients—e.g. phenacemide ("Sedormid").

Purpuric patients bleed because the blood clots poorly, or because the blood capillaries are faulty, or both. The impaired clotting power of the blood is due to a *lack of platelets*—minute bodies, much smaller than red blood cells, normally numbering some 200,000–400,000 per cmm. Platelets are sometimes called *thrombocytes* (meaning clot cells) and shortage of platelets is called thrombocytopenia (penia = poverty).

Idiopathic* Thrombocytopenia

Attacks of bleeding and purpuric rashes occur at intervals, often from childhood onward. Severe bleeding may cause anæmia; cerebral hæmorrhage may occur and may be fatal. As the name suggests, the platelet count is very low in this disease.

Treatment.—*Blood transfusion* may be necessary after a severe hæmorrhage. *Steroids* may arrest the bleeding during an exacerbation. Removal of the spleen (*splenectomy*) produces a marked improvement or even cure in about two thirds of these patients. The operation is, if possible, carried out during a remission (i.e. when the patient is symptom free, or nearly so).

Schonlein-Henoch Syndrome (Anaphylactoid Purpura)

Here there may be three sets of symptoms: joint pains and swellings; abdominal pain, vomiting and hæmorrhage from the bowel; and a rash. Any of these symptoms may be the first to appear. Often the illness follows an acute streptococcal infection.

The *rash* usually affects the back of the forearms and elbows, the shins, and the buttocks and lower part of the back. Urticarial wheals first appear; these develop into raised pink spots which gradually become dark red and purplish. The abdominal symptoms may be so severe as to suggest a surgical emergency and, in fact, intestinal obstruction may result from intense congestion of the bowel wall.

Treatment.—No curative treatment is known. The patient is usually kept in bed during the acute stages. Steroids may relieve symptoms. Immediate operation may be necessary for acute obstruction.

Hereditary Hæmorrhagic Telangiectasia†

In this inherited disorder bright red or purplish slightly raised spots appear, usually on the face, lips, tongue and finger tips. The spots increase in number as the patient grows older. The spots represent groups of abnormally dilated blood vessels which are apt to bleed after slight injury. There may be similar lesions in the mucous membrane lining the nose, respiratory or alimentary tract or the uterus, and these may also bleed. Epistaxis (nose bleeds) are particularly common.

*Idiopathic = of unknown cause, (literally "self-disease"—i.e. self-originating.)

† Telos = end; angion = blood vessel; ectasia = dilatation. Telangiectasia = dilatation of capillaries (end blood vessels).

Treatment.—Often none is required. Superficial telangiectases can be cauterized. After severe bleeding blood transfusion may be required.

Hæmophilia

This is another hereditary disease. It affects only males, but is transmitted by females—i.e. the women in a hæmophilic family are normal, but their sons are bleeders. The hæmophilic suffers from prolonged and excessive bleeding after slight injury—for instance, he may bleed for days after a tooth extraction. Excessive bruising may occur, and one of the most serious results of the disease is the crippling which follows repeated hæmorrhage into the joints.

If hæmophilic blood is drawn off into a test tube it will remain unclotted for a long time. This delayed clotting is the cause of the bleeding. Hæmophilic blood lacks a protein which is present in normal blood and which is essential for normal clotting. The platelets and the capillaries are normal in hæmophilia.

Treatment.—The bleeding may be arrested by transfusion of *fresh blood*, or of *fresh frozen plasma*, either of which supply the missing factor, and temporarily enable the blood to clot normally. A concentrated preparation of the missing factor—*anti-hæmophilic globulin*—is also available, but is at present extremely scarce.

Local Treatment of Wounds in Hæmophilia.—The wound is carefully cleansed and a preparation of *thrombin* is applied. Thrombin is prepared from normal human blood; it enables hæmophilic blood to clot and hence stops the bleeding from a cut or wound.

Christmas Disease (Hæmophilia B)

The symptoms are identical with those of hæmophilia. The missing factor is different, however. Bleeding can be stopped by transfusions of serum* as well as blood or plasma.

Christmas disease is so called because it was first described in a child called Christmas.

(8) Polycythæmia

This is the opposite of anæmia, the blood containing an *excess of red cells*.†

* Plasma: the fluid part of unclotted blood. Serum: the fluid remaining when blood clots.

† Poly—many; cyte—cell; haima—blood.

Polycythaemia vera (Primary polycythaemia)

In this disease, whose cause is unknown, there is a gross over-production of red cells in the bone marrow. The patient is usually a middle-aged man with a rich purplish-red complexion. He may complain of giddiness, breathlessness and fulness in the head. He may have had some form of *haemorrhage* (e.g. nose bleeds, bleeding piles), or of *thrombosis* of veins or arteries (e.g. a cerebral thrombosis). His *blood pressure* is often high; his *spleen* may be enlarged. *Blood count* shows a great excess of red cells—up to 10 million per cmm., and the white cell and platelet counts are increased too.

Treatment.—Frequent venesections are necessary. In addition, the overactivity of the bone marrow can be checked by means of *radioactive phosphorus* or one of the *nitrogen mustard* compounds.

Secondary Polycythaemia.—Whenever there is a chronic severe oxygen lack, the bone marrow responds by producing more red cells. Thus, though each red cell carries less oxygen, there are more red cells in the circulation, so that the total amount of oxygen carried may be the same. This occurs in many forms of chronic lung disease (e.g. emphysema), in cyanotic congenital heart disease, and among people living at high altitudes.

Blood counts often show that the red cell count is raised, though never to the very high levels found in polycythaemia vera.

No treatment of secondary polycythaemia is needed, for, clearly, as the extra red cells are helping to make good the lack of oxygen, we do not want to get rid of them.

(9) Agranulocytosis

This is an acute and dangerous disease, in which the patient has high fever, rigors, and foul, sloughing ulcers of the mouth and throat. He may have been taking a drug such as a sulphonamide (p. 97) amidopyrine (present in many headache powders), or thiouracil (p. 219); or the bone marrow may be diseased, as in aplastic anaemia.

Blood Count.—The patient may be anæmic, but in such cases we are more interested in the *white count*—that is, the number and types of white cells (leucocyte) present in the blood. (For the sake of simplicity and clearness white counts have been omitted from the blood counts given so far, although in actual practice they would usually be included.)

Here is a typical white count from a case of agranulocytosis, compared with a normal one:

Total white blood cells		Agranulocytosis	Normal
		3,000 per c. mm.	8,000
Granulocytes	(Polymorphs (neutrophil)	780 „ „ (26%)	5,600 (70%)
	Eosinophils	0 „ „ —	120 (1.5%)
	Basophils	0 „ „ —	40 (0.5%)
	Lymphocytes	1,590 „ „ (53%)	1,600 (20%)
	Monocytes	630 „ „ (21%)	640 (8%)

Discussion.—From this white count we learn that, in agranulocytosis, the *polymorphs*—normally the commonest variety of white cell—have practically disappeared from the blood.* The *lymphocytes* and *monocytes* are just as numerous as before, and so, of course, make up a larger percentage of the whole.

The absence of polymorphs gives us the clue to the patient's symptoms. Polymorphs are the body's first defence against infection, for they can engulf and destroy invading bacteria. When there are very few polymorphs in the blood any slight infection rages unopposed—hence the high fever and ulcerated throat in agranulocytosis, for the patient cannot even withstand the usually harmless bacterial residents of the mouth.

Polymorphs, like red cells, are manufactured in the bone marrow. The drugs mentioned above depress the functions of the bone marrow in sensitive subjects.

Treatment of Agranulocytosis. The drug, if any, which caused the trouble must, of course, be stopped. Large doses of penicillin are given, and may save life by keeping the infection under control until the bone marrow has recovered enough to produce more polymorphs.

Case History.—Miss C., a typist, aged 24, sent for the doctor one night because she had developed "tonsillitis". Her usual doctor was off duty. The doctor who saw her found her looking poorly; temperature 103°. Septic tonsils and a few ulcers on the gums. The doctor noticed a slight goitre; while she was examining this the patient casually remarked that she took "tablets" for "thyroid trouble". These turned out to be methyl thiouracil. A blood count showed a very low white count (1200 W.B.C. per c.mm., of which only 40 per cent. were polymorphs). She was admitted to hospital, the drug stopped and large doses of penicillin given. She made a rapid recovery and the white count rose to normal. She either had not been told or had not understood that she should call her doctor for any symptom such as sore throat or fever, *explaining that she was having anti-thyroid drugs* should the doctor be unfamiliar with her medical history.

* The rarer granulocytes—eosinophils and basophils—are absent in *agranulocytosis*, which means lack of granulocytes.

A Further Note on the Leucocytes

We have seen what happens when polymorphs disappear from the blood stream and invading armies of bacteria proceed unopposed. In most cases of acute infection—e.g. pneumonia (p. 94), acute appendicitis—the invading bacteria are countered by *large numbers of extra polymorphs*, sent out by the bone marrow to deal with the emergency. In lobar pneumonia, for instance, there may be 20,000 to 30,000 white cells per c.mm. instead of the normal 5,000 to 10,000; the extra whites are all polymorphs. If there is no *leucocytosis*, as this outpouring of leucocytes is called, the outlook for the patient is very bad.

In some infections—for instance, whooping cough—the blood shows an excess of *lymphocytes*. The exact function of these cells is unknown; they do not devour bacteria, but they probably play some part in the defence against infection. Lymphocytes are produced in the lymph glands, not in the bone marrow like red cells and polymorphs. Young children have relatively more lymphocytes in their blood than adults.

(10) The Leukæmias

Leukæmia is a fatal malignant disease of the blood-forming organs. It is becoming commoner; the death rate from leukæmia has risen by 60 per cent. in the last decade. Most malignant diseases are commonest among the elderly; leukæmia may occur at any age and often attacks children. The cause is unknown; it is possible that the disease may be associated in some way with exposure to X-rays, and that the increasing use of X-rays in past years might in part account for the increasing incidence of leukæmia.

In leukæmia—"white blood"—there is a gross and senseless over-production of the *white corpuscles* (leucocytes) which crowd into the blood in various stages of immaturity. Red cell production is inhibited, causing *anæmia*, and *purpura* often results because enough platelets cannot be produced. Although the white count may be a hundred times that of normal blood, so few of these cells are fully formed and mature that the functions of the leucocytes—defence against infection—are also interfered with. Thus *infections* are very common, especially of the mouth and throat where foul, sloughing ulcers may develop. The spleen and lymph glands are enlarged in leukæmia because of the accumulation of immature white cells.

Chronic Myeloid Leukæmia

Here the *polymorphs* are the white cells affected. The patient is usually over the age of 30. The onset is insidious.



Fig 36 Chronic Myeloid Leukæmia

The lines drawn on the abdomen show how the grossly enlarged spleen became smaller under treatment with urethane, the white count fell from 200,000 to 15,000 white cells per c mm at the same time (Dr Richard Asher's case)

The patient easily becomes tired and out of breath. Sometimes his only complaint is of swelling of the abdomen, caused by the spleen which may be enormous in this disease. At times he is feverish; soon he begins to lose weight. Acute exacerbations may occur, with fever and bleeding from the nose and other sites. In the final stages wasting, severe anæmia, fever, hæmorrhages and ulceration of the mouth are seen. Death usually occurs within a few years of diagnosis.

Investigations.—The blood count generally shows an exceedingly high white count; both mature and primitive polymorphs are seen.

Treatment.—The overproduction of white cells can be checked temporarily by means of *drugs* or *X-rays*. The drugs used include busulphan ("myleran") and urethane. Regular blood counts are necessary to make sure that too many white cells (and platelets) are

not destroyed. *Deep X-ray treatment* may be given to the enlarged spleen and to the body generally.

Repeated *blood transfusions* are given to correct anæmia. *Antibiotics* are given for throat and other infections.

It is doubtful if any treatment prolongs life very much, but it can often keep the patient in reasonable health for a considerable time.

• **Chronic Lymphatic Leukæmia**

The patient is usually middle aged; symptoms develop insidiously with anæmia and enlargement of the glands in the neck, axilla or other sites. Acute exacerbations with hæmorrhage may occur. The prognosis is similar to that of chronic myeloid leukæmia.

Investigations.—The white count is usually not so high as in myeloid leukæmia; the excess white cells are *lymphocytes*.

Treatment.—Intravenous injections of nitrogen mustard; or oral preparations of related drugs such as chlorambucil may help, and deep X-ray treatment may relieve symptoms.

Acute Leukæmia

The patient is usually a young child, and the onset of the illness is acute, with fever, severe anæmia and hæmorrhages, sore throat and stomatitis.

Investigations show that the white count is only moderately raised. The great majority of these white cells are extremely immature.

Treatment.—Although the disease is always fatal, worthwhile remissions can be achieved by giving 6-mercapto-purine or aminopterin. Both these drugs are very toxic; treatment must be controlled by frequent blood counts. Blood transfusions and antibiotics are given as described above. Large doses of steroids often cause temporary remissions.

A Note on the Blood

Here is a summary of what we have learnt so far about the blood, with a few additional facts.

The normal adult has about ten pints of blood, comprising some $5\frac{1}{2}$ pints of a nearly colourless fluid called *plasma* and about $4\frac{1}{2}$ pints of *cells*. There are two kinds of cells, red and white. *The function of the red cells* is to carry oxygen all over the body; this oxygen they pick up as the blood circulates through the lungs. The red cells can do this because they contain *hæmoglobin*, which is an oxygen carrier.

The function of the polymorphs—the commonest type of white cell—is to engulf bacteria and so defend the body against infection. The exact function of the *lymphocytes* is not known. *The function of the platelets*—minute bodies much smaller than red or white cells—is to help the blood to clot in the event of an injury in which blood is shed.



Fig 37 Acute Leukemia
Teeth and gums showing hemorrhages
From Fanconi and Wallgren's Textbook of Paediatrics (Heinemann)

Blood Groups.—When a blood transfusion has to be given we need to know the patient's and donor's *blood groups*. There are four main groups—O, A, B and AB. A patient must not be given blood of a group containing a letter A or B absent from his own blood group. Thus, a man whose blood group is A may be given group O or group A blood, but not groups B or AB; the B factor would make the transfused blood *incompatible*.

If incompatible blood is transfused the patient may complain of a sudden *pain in the loin*; fever and rigors are also common. Later the *urine* becomes scanty and blood-stained. This is because the red cells in the incompatible blood rapidly form clumps in the circulation; the clumped cells are then broken down and the debris clogs the kidney tubules and interferes with the formation of urine. Nurses in charge of a patient who is receiving a blood transfusion must keep a sharp look out for any untoward symptoms and report them at once to the doctor. If the transfused blood is found to be incompatible the transfusion will have to be stopped at once. If

there is any question of incompatibility of the blood *keep the bottle of blood*; it will be needed for investigation. N.B.—*Dirty apparatus* will cause symptoms very like those of an incompatible blood transfusion.

The importance of another factor—the *Rh factor*—is now well recognised. Eighty-five per cent. of the population have this factor in their blood. The remaining 15 per cent. are *Rh negative*. If several transfusions of Rh positive blood are given to an Rh negative patient he may become *sensitised* to the Rh factor, and transfusion reactions just like those described above may then occur. This is especially serious in women patients. If a woman is Rh negative and her husband is Rh positive her baby is likely to be Rh positive too. If her blood has been sensitised by Rh positive blood transfusions it may antagonise the baby's blood, and the child may be born with a severe—often fatal—blood disease. If she has several children the later babies may be affected even if she has had no blood transfusions, the earlier pregnancies having sensitised her to Rh positive blood (see p. 383).

Classification of the Anæmias

- (1) Anæmia due to loss of blood (including anæmia in bleeding diseases).
- (2) Anæmia due to faulty red cell production :
 (the dyshæmopoietic* anæmias) Iron deficiency anæmia. Pernicious anæmia. Other macrocytic anæmias. Aplastic anæmia. Leukæmia. Anæmia in many infections, renal failure etc. (and see below).
- (3) Anæmia due to hæmolysis (destruction of red cells in circulation). Congenital hæmolytic anæmia. Hæmolytic anæmia in malaria, septicæmia and many toxic conditions.
 (N.B.—Anæmia in certain infections and toxic conditions may be caused both by faulty production and hæmolysis of red cells.)

(11) Diseases of the Lymphatic Glands

- (a) The commonest disease affecting the lymphatic glands is *acute inflammation* (lymphadenitis). Inflamed lymph glands are swollen and tender. Lymphadenitis generally accompanies some form of local sepsis—for instance, the glands in the neck are swollen in tonsillitis, and a septic finger may cause enlargement of the glands

*Dyshæmopoietic. Dys—difficult; hæma—blood; poiesis—production.

in the armpit. This is because the lymph glands act as bacterial traps which stop the infection invading the blood stream. The inflammation in the glands may go on to *suppuration*. The *treatment* of acute lymphadenitis is that of the cause; if pus forms incision will be necessary.

Lymphadenitis also occurs in many fevers, for instance rubella (p. 336), diphtheria (p. 328) and syphilis (p. 347). It is one of the main symptoms of glandular fever (p. 345).

(b) In *cancer* malignant cells often spread to the local lymph glands—for instance, the axillary glands are often involved in cancer of the breast. This tells the surgeon that the outlook for the patient is poor, for it will be difficult to remove every trace of malignant tissue at operation.

(c) *Tuberculous Adenitis*.—This is much less common than formerly, but cases are still seen from time to time, especially in the country, where unpasteurised milk is still sometimes drunk. This is because tuberculous adenitis is often caused by drinking infected milk from a tuberculous cow (p. 109). It may also be caused by infection from a human source. Tubercle bacilli usually reach the glands in the neck via the tonsil, though other routes are possible.

The patient with tuberculous adenitis is usually a child. The glands on one side of the neck are most often affected. The glands are swollen, and, if untreated, soon become fixed to skin, muscle and other structures. The centre of the gland may liquify, so that the swelling fluctuates on pressure, but the overlying skin is neither hot nor red as it would be in acute septic lymphadenitis. For this reason a tuberculous abscess is often called a *cold abscess*.

Tubercle bacilli can often be found in material aspirated from the gland. The *tuberculin skin test* is positive. The *X-ray of chest* is usually normal.

Treatment.—Anti-tuberculous chemotherapy for one to two years is the basis of treatment. The patient may be admitted to hospital or sanatorium for the first part of the treatment. *Surgery* is necessary in some cases—either to drain a cold abscess or to excise tuberculous glands. The results of modern treatment are excellent. If treatment is delayed, the abscess may discharge through the skin, causing a disfiguring scar. The infection may spread, via the lymphatics or blood stream, to other parts of the body, but this is rare after adequate chemotherapy.

A patient with tuberculous adenitis must be notified to the Medical Officer of Health, and contacts are examined as in pulmonary tuberculosis. Supervision is continued for at least five years.

It is interesting to remember that tuberculous adenitis (scrofula) was once known as "the King's Evil" as it was supposed to be cured by the King's touch. Samuel Pepys on June 23, 1660, went "to see the King [Charles II] touch people for the King's Evil. But he did not come at all, it rayned so; and the poor people were forced to stand all the morning in the rain in the garden. Afterward he touched them in the Banquetting-house." Pepys does not report the results of this treatment.

Hodgkin's Disease

This is a fatal disease of unknown cause, involving lymph glands, spleen, liver and lymphatic tissue all over the body. The disease may come on at any age, though it is rare below the age of 20. The glands in the neck are often the first affected, and the patient consults his doctor because he has noticed a group of firm rubbery lumps on one or both sides of the neck. Later the glands in the axillæ and groins may be involved, the spleen and liver enlarge, and glandular masses in the chest and abdomen may cause varied symptoms by compressing vital structures.

General symptoms include loss of weight, anæmia, irregular fever, itching and, in some cases, discoloration of the skin. Purpuric rashes and hæmorrhages may occur in the late stages.

Investigations.—*X-ray of the chest* may show enlarged glands in the mediastinum. *Blood counts* usually show some anæmia, and white cells and platelets may also be reduced in numbers. *Biopsy.* This means the removal of tissue for microscopic examination. A gland is excised under local anæsthesia and sections are examined under the microscope. Biopsy is usually necessary to distinguish Hodgkin's disease from lymphosarcoma and other diseases causing enlargement of the lymph glands.

Treatment.—No treatment is known which will cure Hodgkin's disease. Probably no treatment prolongs life, though striking temporary improvement may result. *Deep X-rays* to the spleen and affected glands often produces remissions lasting weeks, months or occasionally years. The glandular masses shrink and the general condition improves. *Nitrogen mustard, chlorambucil* and related drugs may be given as well as X-ray treatment, or after the radiotherapy has ceased to be effective. They may produce worth-while remissions, but are very toxic.

Prognosis.—Hodgkin's disease is always fatal, usually in three or four years.

Lymphosarcoma

This is a malignant disease affecting lymph glands, spleen and lymphatic tissues throughout the body. The symptoms are very similar to those of Hodgkin's disease; the two diseases can usually only be distinguished by means of biopsy. Lymphosarcoma is even more rapidly fatal, the patient dying, as a rule, in about two years. The *treatment* is similar to that of Hodgkin's disease.

DIGESTIVE DISORDERS

(1) Peptic Ulcer (Gastric and Duodenal Ulcer)

IN a modern, civilised community peptic ulcer is by far the most important digestive disorder. It has become very much commoner during the past fifty years; perhaps one in ten of the population suffer from peptic ulcer at one time or another. Many a large hospital has a whole ward devoted to the treatment of ulcer patients, and many thousands of sufferers who are never admitted to hospital are treated by their family doctors or in Out-Patients.

Cause of peptic ulcer.—This is largely unknown. The striking rise in incidence, especially of duodenal ulcer, suggests that our way of life or our diet is in some way responsible. Relapses are often precipitated by worry and overwork.

Peptic ulcers only occur where *gastric juice* can penetrate—in the stomach itself, and in the first part of the duodenum (which leads out of the stomach); or in the portion of small intestine which has been joined on to the stomach at operation (anastomotic ulcer); rarely, at the lower end of the oesophagus.

Although the symptoms and treatment of gastric and duodenal ulcer are very similar they may not be caused in the same way. Duodenal ulcer is much the commoner of the two. Duodenal ulcer affects men much more often than women, and attacks all classes equally. Gastric ulcer is only slightly commoner among men than women; it is rare among the rich and common among the working class.

Curiously enough, peptic ulcer of both types is more common among people whose blood is group O than among those with other groups.

Symptoms.—The patient is usually a middle-aged man. He will describe how he has suffered from indigestion on and off for years, but only recently has it been severe enough to trouble him much. About half an hour after a meal he is seized by a sharp, burning pain “just here”—and he puts the tips of his fingers to the pit of his stomach, just below the breast bone. The pain lasts from a few minutes to an hour; sometimes it makes him vomit, and this stops the pain; alkaline powders also give relief. Once or twice he

has vomited blood, and on these occasions he noticed that his stools were very dark for a few days. His appetite is good—he would like to eat, but dare not for fear of the pain, and so he has lost weight.



Fig. 38. Gastric Ulcer.

X-ray of stomach following barium meal. The ulcer is on the lesser curvature (the left-hand border in the picture). (Dr. Avery Jones' case. X-rays by Dr. Pygott.)

Sometimes he enjoys months of freedom from indigestion when he can eat what he likes without bringing on the pain. He may remember that his last bad attack followed some such incident as a row with the boss and the threat of losing his job.

Such a history is almost enough in itself to make the diagnosis of *gastric ulcer*. In *duodenal ulcer* the pain occurs two or three hours after food, instead of just after the meal, and it is generally *relieved* if the patient eats something. It often awakes the patient at night. Vomiting and the vomiting of blood (*hæmatemesis*) may occur, and altered blood often turns the stools black and tarry (*melæna*). As in gastric ulcer the patient often goes for months without suffering any pain, and these pain-free periods so often occur during his holidays, while relapses coincide with bouts of overwork at the office that many of these men were, in the past, dubbed work-shy neurotics.



Fig. 39. Stomach opened to show large chronic gastric ulcer.

Investigation of a Case of Peptic Ulcer

Barium Meal.—An *X-ray* is necessary in every case, both to confirm the diagnosis and so that the progress of the ulcer may be watched in later films. The stomach and duodenum show up in an *X-ray* after the patient has swallowed an opaque mixture of barium or bismuth. The ulcer itself can usually be seen in the *X-ray* film. The actual movements of the stomach can be watched on the *X-ray* screen. The movements of the stomach are usually extra vigorous in these patients.

Gastric Analysis.—Samples of gastric juice can be withdrawn by means of a narrow rubber tube (Ryle's tube) which the patient

swallows, leaving one end hanging out. The stomach is emptied by means of a syringe and its contents are sent to the laboratory for analysis. There is usually too much *hydrochloric acid* in the gastric juice of a duodenal ulcer patient. (The gastric juice is often *normal* in a case of gastric ulcer.)

For a *test meal* the patient swallows some gruel, of which samples are withdrawn every half-hour and analysed. In a case of duodenal ulcer the gruel is often digested with unusual speed and the stomach quickly empties itself (i.e. in less than the usual $2\frac{1}{2}$ hours). *Alcohol* may be given instead of gruel; it is a stronger stimulus to acid production.

Cause of the Symptoms

The *pain* in peptic ulcer is probably largely due to the action of the *acid gastric juice* on the ulcer. Strong contractions of the stomach may also cause pain.

The ulcer may repeatedly heal and then break down again; this accounts for the alternating bouts of indigestion and freedom from pain so frequently seen. The fact that worry and overwork so often bring on a relapse is not surprising, for the stomach is much influenced by the mind and the emotions. (Most of us have felt sick with anxiety, or lost our appetites at times of emotional stress, or experienced gastric discomforts of one sort or another at such times.)

Bleeding may occur from any ulcer; a severe hæmorrhage occurs when the ulcer eats away the wall of a large artery. A bleeding ulcer may cause a hæmatemesis, or the blood may pass down the intestine, appearing, in altered form, in the tarry melæna stool. A hæmatemesis is often followed by melæna.

The Treatment of Peptic Ulcer

We have seen that ulcer symptoms are made worse by overwork and worry; we know, too, that many of these patients have over-active stomachs and a highly acid gastric juice which irritates the ulcer, causes pain and prevents healing. So the patient needs rest for mind, body and stomach, and he needs something to counteract the acidity of the gastric juice if the ulcer is to be allowed to heal.

A patient with a severe ulcer needs a period of rest in bed, with sedatives, such as phenobarbitone, if he is mentally upset. At the same time his stomach is rested by restricting the diet to milk—easily digested and non-irritating—and milk preparations such as cocoa, invalid foods, egg custard and junket. If milk is his only food the patient will need about four pints a day, given in hourly or two-

hourly feeds to avoid overloading the stomach at any one time. It is even more effective to give the milk day and night by a continuous gastric drip. This may be kept up for several days by means of an indwelling Ryle's tube. The tube is removed daily for cleansing. Small meals may be allowed as well as the milk drip. Extra vitamin B is given in the form of yeast extract, and vitamin C as ascorbic acid (50 m.gm. three times a day), for these vitamins are scanty in milk.

With small, frequent feeds the stomach is never long empty and so the ulcer is spared the unopposed action of the acid gastric juice.

The acidity of the gastric juice can be counteracted by giving *alkalis* such as magnesium trisilicate or aluminium hydroxide in teaspoonful doses every hour or two at first, then less frequently. Alkalis generally relieve the pain rapidly.

The patient's diet is gradually increased as he improves, and bread and butter, potatoes, sieved vegetables and fruit, fish, eggs and, finally, meat, are added. Strongly spiced foods, meat soups, alcohol and tobacco are forbidden while he is in hospital, for they irritate the stomach and increase the flow of gastric juice.

The patient should be taught to chew his food well, and he should have his teeth seen to while in hospital—he cannot chew if he needs dentures or has toothache.

Surgical Treatment.—Although the symptoms of peptic ulcer rapidly respond to the treatment outlined above, and X-rays will show that the ulcer has healed, relapse is all too common. After several disabling relapses surgery may be considered. The operation usually advised is *partial gastrectomy* (removal of part of the stomach). Although this is a severe operation, if it is performed by an expert team the mortality is low and the results are often excellent.

An operation which is less frequently performed is *gastroenterostomy* (by-passing the ulcer by joining a loop of small intestine on to the stomach). Gastroenterostomy may be combined with *vagotomy* (cutting the vagus nerve, which stimulates gastric motility and gastric juice secretion).

Complications of Surgery.—(1) An *anastomotic ulcer* may develop at or near the junction of the small intestine with the stomach.

(2) *Dumping Syndrome.*—Soon after a meal the patient feels weak, giddy and faint, flushes and sweats profusely. The symptoms may be due to the rapid emptying of the remaining portion of the stomach into the small intestine, causing reflex disturbances; or they may be due to disturbances of sugar absorption. Symptoms are relieved if the patient takes small frequent meals, including plenty of protein,

lies down after taking food, and drinks between rather than at meals.

Many patients suffer mild symptoms of "dumping" after gastrectomy or gastroenterostomy, but fortunately they are rarely disabling.

After-care.—On discharge from hospital, whether the patient has had medical or surgical treatment, he must be advised to stick to regular hours, eat small frequent meals and avoid anything he has found to bring on the indigestion. What he eats is much less important than when and how he eats it. He must never go more than $2\frac{1}{2}$ hours without food, and should take a glass of milk or some biscuits between his main meals. Alcohol and tobacco on an empty stomach must be forbidden. If his job involves irregular hours, heavy strain or shift work he should change it, though this, unfortunately, is often impossible. He may take alkalis if the pain recurs, but he should always report any relapse to the doctor.

Complications of Peptic Ulcer

Hæmorrhage.—Slight bleeding from a peptic ulcer is common and needs no special treatment except for a short period of rest in bed. A massive hæmorrhage is dangerous, and may be fatal, especially in older patients. *Suspect bleeding* if an ulcer patient suddenly becomes pale, restless and clammy, turning his head from side to side, smacking his dry lips and asking for repeated drinks of water. Report any such symptoms to the doctor immediately, keep the patient quiet and warm with the head low, and take the pulse every $\frac{1}{4}$ – $\frac{1}{2}$ hour. A feeble pulse and a rising pulse rate are signs suggestive of severe bleeding. The blood may be vomited at once, or a melæna stool may be passed several hours later.

Treatment.—If a sedative is necessary, an injection of soluble phenobarbitone (gr. 3) may be given (or morphia—but this is apt to cause vomiting). If the patient is well enough to drink he may be given as much one-third normal saline, flavoured with fruit juice, as he wants, and a jug of such a fluid may be left by his bedside for the first few days. If he cannot drink, fluid must be given by rectal or intravenous infusion. A blood transfusion will be necessary in all severe cases.

As a rule the patient may be allowed two-hourly feeds of puréed food from the beginning, or of milk (7 ounces) if he prefers. Occasionally absolute starvation is advised. Intravenous fluids will then be necessary. The patient is usually kept in bed for 10–14 days after the bleeding has stopped; after which he will need a period of convalescence on an ulcer diet.

Severe continued or repeated bleeding from an ulcer is often treated surgically. Large quantities of blood are transfused, to replace what the patient has lost and is losing, and the transfusion is kept running throughout the operation (partial gastrectomy).

Pyloric Stenosis.—This means narrowing of the opening from the stomach into the duodenum—the *pylorus*. It may occur when a great deal of scar tissue develops in a pyloric ulcer. Food cannot pass out of the stomach normally, and the patient suffers from repeated attacks of copious vomiting, often bringing up remains of food eaten hours or even days before. Intensive ulcer treatment combined with washouts of the dilated stomach may relieve such a patient, but an operation is often necessary. Intravenous fluids will be necessary if the patient is dehydrated or suffering from loss of electrolytes.

Perforation is the most serious complication of peptic ulcer; it occurs when an ulcer eats its way through the wall of the stomach or duodenum. If the perforation occurs soon after a meal undigested food may gush out and flood the peritoneum. The patient is seized by a sudden violent pain in the upper abdomen and quickly becomes prostrated as peritonitis develops. Immediate operation is the usual treatment. If, however, symptoms suggest that the leak is a small one, or if the patient would not stand operation, *medical treatment* may be advised. The stomach is kept empty by continuous gastric suction, intravenous fluids are given to correct dehydration and loss of electrolytes, and antibiotics to prevent infection.

Prognosis in Peptic Ulcer

Although the patient may recover completely and permanently after one or more courses of medical treatment it is impossible to be certain that he will never relapse; even after partial gastrectomy an anastomotic ulcer may occur. Hæmatemesis or perforation may occur in any peptic ulcer patient, and may be fatal. The total death rate, however, is very low, considering the enormous numbers of ulcer patients in the community.

Case Histories

Mr. L., aged 49, a builder.—He had had indigestion on and off for 7 years. Six years before he had had a perforation which was operated on. For the past year he had had attacks of vomiting which had become more and more severe and frequent; he had twice vomited a small amount of blood. He was an aggressive, uncooperative man who would not obey any instructions one gave him. He had repeated spells in bed at home and in hospital; his stomach was grossly distended, as shown by X-ray, and anything he ate was held up for hours before it could pass the obstruc-

tion at the pylorus. Recently he was admitted to hospital and a gastro-enterostomy performed. At present he is working and seems to be doing well, but he has the sort of temperament which makes relapses likely.

Mr. B., aged 42, a lawyer.—He has had duodenal ulcer symptoms on and off for many years. He has had three courses of medical treatment in hospital and several spells in bed at home. He is careful with his diet, keeps regular hours and takes alkali powders when he gets attacks of pain. He manages to keep his symptoms at bay and to live an active life without suffering unduly.

Mr. H., a surgeon, aged 39, first had gastric ulcer symptoms 8 years ago. Six years ago he had a severe hæmatemesis and was warned to take things more easily. He ignored all diet sheets and restrictions and during the war did two or three men's work. However, at length symptoms became so severe that a gastrectomy was necessary. A very large ulcer was found at operation. Since the operation he has been well.

A Note on Vomiting

Sickness is a common symptom of stomach disorders and also of many other conditions. As we have seen, vomiting occurs in peptic ulcer. The gastric irritation caused by bacterial food poisoning (p. 321) or by chemical poisons, including alcohol, causes severe vomiting. Patients with heart failure are often sick because of the congestion of the stomach.

Many other painful abdominal conditions cause vomiting—cholecystitis (p. 172), appendicitis, etc. Acute intestinal obstruction causes severe and intractable vomiting.

Many toxic conditions such as uræmia (p. 198), diabetes (p. 246) and digitalis poisoning (p. 53) cause vomiting, although the digestive tract is not itself diseased. Vomiting is common at the onset of many fevers, especially in children. Pregnant women are often sick; the exact cause of pregnancy vomiting is not known.

Curiously enough, disturbances of the central nervous system often cause vomiting. Migraine (p. 313) is nearly always associated with sickness. Seasickness is caused by an upset of the balancing mechanism of the brain. Brain tumours (p. 300), meningitis (p. 323) and other diseases which raise the pressure inside the skull often cause severe vomiting. Finally, vomiting may have psychological causes—e.g. disgusting sights or smells may make a person sick, and some hysterics vomit for very little cause.

The nature of the vomited material (vomit). Always notice what the vomitus is like, and save it for the doctor's inspection if necessary. Vomitus usually consists of a mixture of digestive juices with partially digested food. *Vomiting of blood* is commonly a symptom of peptic ulcer (p. 169), but also occurs in cancer of the stomach and certain

blood diseases. In pyloric obstruction (p. 170) the vomitus is very copious, and in it may be recognised food eaten days before.

The *treatment* of vomiting is that of the cause. When this is some gastric irritant, such as alcohol, it is often helpful to empty the stomach with a tube and wash it out with saline or sodium bicarbonate solution (two teaspoonfuls to the pint). Travel sickness may respond to belladonna preparations or anti-histamines such as dimenhydrinate ("Dramamine") or meclizine ("Sea legs", "Bonamine"). These drugs may also be given in pregnancy sickness. A patient who has been sick repeatedly may become depleted of fluid, especially if he also has diarrhœa, and saline infusions may be necessary.

(2) Cholecystitis and Gall Stones

Cholecystitis, or inflammation of the gall bladder, is common among well-fed middle-aged women (it is much less common among men).

Cholecystitis and gall stones are very often found together; often it is hard to say which is cause and which is effect. The diet, which affects the composition of the bile, probably plays an important part in the causation of cholecystitis and gall stones; a fatty diet is probably harmful.

Acute Cholecystitis.—This is either associated with gall stones, or with an acute infection such as typhoid. The patient is often very ill, with fever, pain in the right upper abdomen and vomiting.

Treatment.—Bed rest, hot water bottles applied to the abdomen, sedative and pain relieving drugs, and antibiotics are prescribed. Surgery is avoided during the acute stage whenever possible.

Chronic Cholecystitis.—The patient suffers from a dull aching pain, or a vague sensation of fulness in the upper abdomen. She also has a poor appetite—especially for fatty foods.

Symptoms of Gall Stones.—Often there are none; gall stones are quite common among apparently healthy people. In many cases, however, gall stones cause the symptoms of cholecystitis (see above) or gall stone colic.

Gall-stone "Colic".—The patient suffers from attacks of pain, which is often agonisingly severe. The pain is felt below the right ribs, and often passes through to the back and up to the right shoulder. The pain rises to a crescendo, continues for a period of minutes or hours, and then gradually dies away. (It is not usually a colicky, spasmodic, pain.) After the attack the patient may be slightly jaundiced, and the urine may be dark and the stools pale. Repeated attacks of pain may occur.

Investigation.—Gall stones may or may not show up in a straight X-ray of the abdomen. In suspected gall bladder disease a *cholecystogram* is often called for. The patient is given overnight a dose of a special salt (tetraiodophenol phthalein) which collects in the bile and shows up on X-ray. X-rays are taken in the morning, before and after a fatty meal (fat stimulates the gall bladder to empty itself).

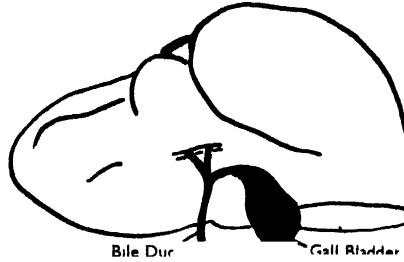


Fig 40 Diagram of the Liver, Gall Bladder and Bile Ducts, seen from behind



Fig. 41. Normal Cholecystogram

In cholecystitis the gall bladder either does not fill at all, or fills slowly and irregularly and empties slowly.

A cholecystogram cannot be done if the patient is jaundiced.

Cause of the Symptoms.—The gall bladder is a reservoir where bile is stored and concentrated on its way from the liver to the duodenum.

Bile is necessary for the proper absorption of fat in the food. An irregular supply of bile often causes indigestion when fatty food is eaten. *Gall-stone colic* occurs when a stone becomes impacted in the duct through which bile passes from the gall bladder to the duodenum. If the bile duct is blocked by the stone for any length of time, jaundice, dark urine and pale stools occur (see p. 177).

Treatment.—The patient should avoid eggs, butter, cream, pork and fried foods. Preparations of *bile salts* may relieve the dyspepsia. Attacks of gall stone colic are treated with morphia or pethidine, and, in some cases amyl nitrite or nitroglycerine to relieve the spasm of the bile duct and allow the stone to pass.

When gall stones are present and are causing symptoms operation (cholecystectomy, or removal of the gall bladder) is usually advised.

(3) Infective Hepatitis (Epidemic Jaundice)

The largest gland in the body is the liver, and, as it pours its secretion (bile) into the digestive tract, liver disorders may cause indigestion. The liver, however, has many other functions besides the production of bile so that the symptoms of liver disease are many and various.

In this country the commonest liver disease is *infective hepatitis*. This is an infection which may occur in epidemics, especially among children and young adults; it was very common during the second world war. The infecting organism is a *virus*—an organism smaller than bacteria—which is swallowed; virus is present in the stools during the early stages of the illness. The incubation period is 3–5 weeks.

Symptoms.—The first symptom is generally a complete loss of appetite amounting to a positive loathing for food—"The thought of food makes me shudder" as one sufferer put it. Nausea and vomiting are common; there may be some abdominal discomfort and aching, especially under the right ribs. The temperature soon rises and the patient may feel very ill for a few days. During this time the urine darkens and the stools become pale and greasy.

Jaundice appears within 3–6 days of the onset of symptoms. It may be limited to a slight lemon tinting of the whites of the eyes or the patient may turn a deep buttercup yellow. He feels better at this stage, and the fever and other symptoms abate, though a poor appetite and depression—"the jaundiced outlook"—are common throughout the disease. Jaundice may persist for a few days or several weeks. The illness, having run its course, generally clears up completely. Relapses are uncommon and death an extreme rarity.

In *mild cases* there may be no pain, fever or jaundice, the only symptoms being a digestive upset, dark urine and pale stools. Mild cases often outnumber severe during an epidemic.

The cause of the symptoms is an acute inflammation of the liver. As we have seen, one of the functions of this organ is to secrete bile, in which is discharged the body's excess of the yellow *bilirubin*. In hepatitis the inflamed liver fails in this task, and bilirubin accumulates in the blood, causing *jaundice*. Some of the accumulated pigment escapes into the urine, which thus becomes darker. As no bile reaches the bowel from the diseased liver the stools are *pale*, for their normal colour derives from the bile. The *loss of appetite*, too, is partly due to the suppression of bile, for this fluid helps in the absorption of the fat in the food. Some jaundiced patients have a particular dislike of fat.

Treatment.—No curative treatment is known. The patient should be kept in bed until the jaundice begins to abate. Starvation is the liver's great enemy, and if it is to recover its health, the inflamed liver must at all costs be supplied with enough *sugar* and also *protein*. If the patient vomits or refuses his meals he must be given intravenous or rectal infusions of glucose. But it is much better, and nearly always possible to overcome his lack of appetite by serving up tempting, well set out meals. The diet need not be fat free, but greasy food should not be served. "A little of what you fancy does you good" applies to the feeding of jaundiced patients—and many others.

As infective hepatitis is an infectious disease, and the organism is present in the stools, barrier nursing precautions are observed in the early infectious stages of the illness.

The patient will need a long convalescence, during which he should be teetotal, for the liver will not yet be fit to stand up to a liver poison such as alcohol.

Prognosis.—The great majority of patients with hepatitis recover completely. Rarely, however, the jaundice deepens and the patient sinks into coma and dies (acute yellow atrophy, acute hepatic necrosis) or he may gradually go downhill with relapsing jaundice (subacute hepatic necrosis). Such cases may go on to cirrhosis of the liver.

Homologous Serum Jaundice. An attack of jaundice indistinguishable from infective hepatitis may follow the transfusion of blood, serum or plasma or the injection of any preparation containing human blood products, such as convalescent serum (p. 298). Jaundice may also occur if any injections are given to a series of patients, or if

samples of blood are taken from several patients, without using a fresh sterile needle *and syringe* for each one. Outbreaks of jaundice in venereal disease clinics used to be due to this cause. The jaundice usually occurs about two to five months after the injection or transfusion, when the patient has probably forgotten all about it. It is thought that the virus of infective hepatitis, or one very like it, must be present in the blood of the donor; even the minute quantities of contaminating blood when a series of injections are given with the same syringe may be enough to spread the disease.

Serum jaundice is a severe, sometimes fatal disease, so everything possible must be done to prevent it. If a series of patients have to be injected—say with penicillin—a fresh sterile needle and syringe must be used for each injection. Syringe and needle may be sterilised by boiling for five minutes, or by autoclaving.

(4) Cirrhosis of the Liver

We have already seen how the liver may be attacked by the virus of infective hepatitis. Acute hepatitis may also be caused by chemical poisons such as arsenic, phosphorus and chloroform. Hepatitis, whatever the cause, may clear up completely, but a small proportion of patients go on to develop cirrhosis of the liver. *Malnutrition* may also cause cirrhosis. Formerly *alcoholism* was thought to be the chief cause of cirrhosis, but in this country at the present time, alcoholic cirrhosis is uncommon, the cost of the necessary spirits being prohibitive. In many cases no cause can be found.

Symptoms.—The patient feels unwell, goes off his food and loses weight; he suffers from dyspepsia, often with pain under the right ribs and belching; his complexion is muddy, and at times he may be somewhat jaundiced. Later on he may bleed from the nose and gums and small dilated veins appear on his face; swelling of the abdomen due to *ascites* is a grave symptom and so is the *vomiting of blood*. Mental confusion is common in the later stages. Finally the patient sinks into coma and dies.

* *Cause of the Symptoms.*—These are the result of the disorganisation of the liver. Dyspepsia and jaundice are due to the interference with the secretion of *bile* (p. 173). *Purpuric symptoms* occur because the liver is concerned with the normal clotting of blood. Other symptoms are the result of the obstruction to the flow of blood through the scarred liver. The blood from the stomach and intestines normally passes through the liver via the *portal venous system*. *Portal obstruction*, such as occurs in cirrhosis, means that the blood

is dammed back in the abdominal organs, and *ascites* develops, just as *œdema* occurs when the veins are overloaded in heart failure. *Hæmatemesis* occurs when the blood from the congested stomach is diverted into the veins of the œsophagus, which may become distended and rupture. *Coma* is due to poisoning with the breakdown products of protein, which are normally dealt with by the liver.

Treatment.—The patient must eat regular meals, with plenty of fruit and vegetables, protein and carbohydrate and not much fat. Vitamin B concentrates are given, and alcohol is forbidden. Should *œdema* and *ascites* appear the patient should be admitted to hospital, and the salt in his diet rigidly restricted. Diuretics, such as chlorothiazide or mersalyl, are given. (Paracentesis is now less used than formerly.) If mental symptoms appear and coma threatens, the high protein diet is stopped at once; *all protein foods are forbidden*, for, at this stage, they are no longer body-builders but potential poisoners. A high carbohydrate diet is given instead, with extra vitamin B by injection. If the patient is in coma, tube feeds and/or intravenous infusions are given.

Prognosis. This is fairly good in the early stages, provided that the patient keeps to his diet. For the patient with *ascites* or *hæmatemesis* the outlook is grave.

A Note on Jaundice

In various illnesses, besides liver disease, the patient may become yellow or *jaundiced*. Jaundice is first noticeable in the whites of the eyes; later the rest of the skin turns yellow and, in severe cases, the patient may finally turn dark brown with a sort of greenish tinge.

The jaundiced patient often has pale stools and dark brown urine. The dark colour of the urine may be noticeable a day or two before the patient himself turns yellow.

Jaundiced patients are apt to be depressed and miserable—"the jaundiced outlook"—and sometimes they are tormented by unbearable itching. In severe cases there is a tendency to bleed unduly, and slight injuries may cause severe hæmorrhage. *Purpuric rashes* are caused by hæmorrhage under the skin.

As we have seen jaundice is due to an accumulation in the blood of the yellow dye *bilirubin*. Bilirubin is produced during the normal breakdown of superannuated red blood cells; it is then excreted by the liver, which passes it into the bile. Bile is stored in the gall bladder and finally reaches the gut via the bile duct.

Thus jaundice may occur when red cells are broken down too fast,

as in haemolytic anæmia (p. 150), so that *too much bilirubin is manufactured*. Or the patient may turn yellow because the *bilirubin is dammed back* by a *diseased liver*, which fails to pass it into the bile. Some of the dammed-back bilirubin escapes into the urine, turning

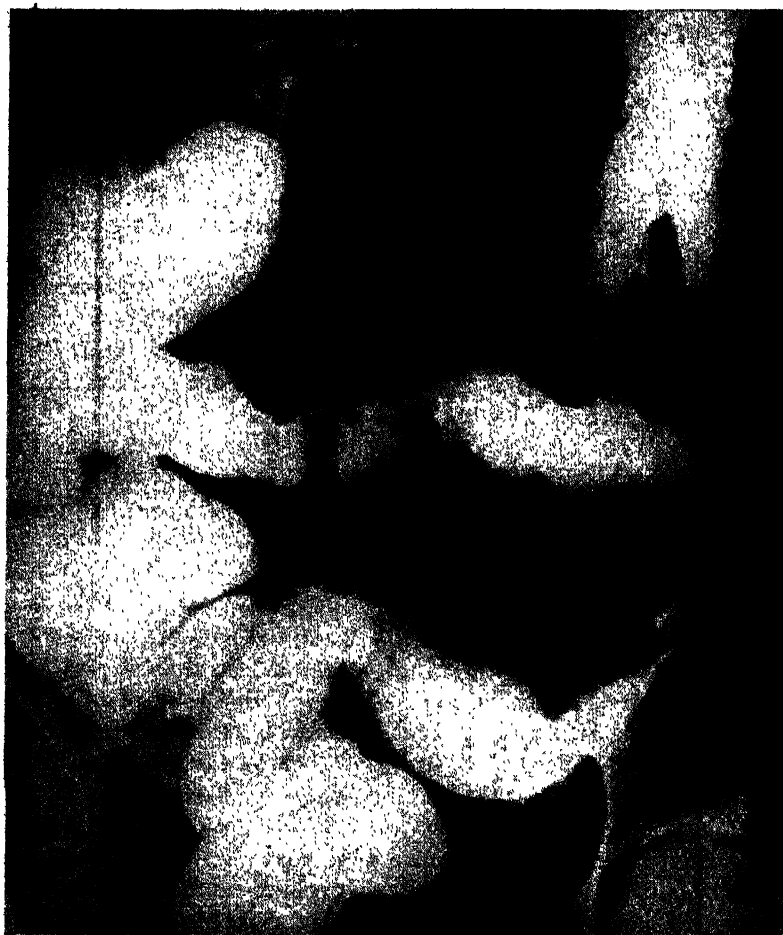


Fig. 42. Ulcerative Colon.

*Barium enema showing absence of normal haustrations (indentations) of the transverse and descending colon and marked narrowing of the descending colon. The ascending colon is dilated. (Compare the ascending colon in Fig. 43, which shows normal haustrations.)

it brown, whereas there is not enough in the bile to give the stools their normal colour, so that they turn a pale grey. Bile helps the absorption of fats from the food; in the absence of bile fat cannot be properly absorbed and passes down the gut and out with the

stools. This extra fat also helps to give the stools of the jaundiced patient their greyish colour.

The deepest jaundice is seen when the *bile is dammed back* by some blockage which prevents its passing into the duodenum. The obstruction may be a gallstone in the bile duct, or a cancer which presses on the duct from outside—often a cancer of the pancreas. In *obstructive jaundice*, as it is called, the patient is apt to suffer from deep jaundice, spontaneous bleeding, frightful itching, and his stools will be nearly white while the urine is the colour of tea.

The *treatment* of jaundice is that of the cause. The *diet* should be easily digestible, and appetising—give the patient whatever he fancies, but see that he gets plenty of sugar, which helps to protect the liver from further damage. *Itching* may be relieved by calamine or lead lotion, with a sedative such as phenobarbitone. The *tendency to bleed* is treated by giving the patient *vitamin K* or one of its preparations; 10 mgm. three times a day may be given, preferably by injection. Large doses must be given before operating on a jaundiced patient, or bleeding at operation may be severe.

(5) Colitis

Colitis, or inflammation of the colon, may be caused by various bacteria, such as those of dysentery and bacterial food poisoning. In what the public call colitis (*mucous colitis*) the colon is not inflamed at all, but is constantly irritated by strong purges and unsuitable food. The patient, who is generally very neurotic about his bowel, passes strings of slimy mucus with his stools. The treatment is that of the associated constipation, indigestion and neurosis.

Ulcerative colitis is a very severe disease of unknown cause. The colon is inflamed, but we do not know what causes the inflammation—it does not seem to be due to any of the well-known bowel infections, such as dysentery. The symptoms are often brought on by emotional upsets. Diet does not appear to have much to do with the causation of ulcerative colitis.

The disease usually comes on gradually, with diarrhœa and blood in the stools. This may continue for years, or the patient may have periods of well-being alternating with weeks or months of diarrhœa. Vomiting and abdominal pain are uncommon. In severe cases the onset is often sudden, with the daily passage of 15 to 20 stools consisting largely of blood and pus. The patient is ill and feverish, and rapidly wastes and becomes anæmic. He may become a chronic invalid or die of exhaustion. *Complications* include perforation of the bowel, intestinal obstruction and cancer.

These symptoms are readily understood when the lower part of the bowel is inspected by means of a *sigmoidoscope*. The mucous membrane lining the bowel is red, swollen and covered with blood and pus. Later on spreading ulcers may strip the bowel of most of its lining; finally scar tissue may develop as the ulcers heal, which may obstruct the bowel. Or an ulcer may perforate, or malignant change may occur.

Treatment.—The patient should be kept in bed during the acute stages.

The *diet* is one which excludes anything which might scratch or irritate the colon, such as fruit pips and skins, stringy vegetables, coarse oatmeal and wholemeal bread. Apart from these the patient may eat what he likes—he needs plenty of nourishment and there is nothing wrong with the rest of his digestive apparatus. Vitamin concentrates are given, sometimes by injection.

If he is anæmic a blood transfusion will often be necessary, and may be followed by a striking improvement in the patient's general condition.

Drugs.—For mild or moderate diarrhoea mixtures of *kaolin*, or *kaolin and pectin*, may be helpful. *Morphia* and *opium* check severe diarrhoea, but can only be given for short periods for fear of causing addiction. *Belladonna* or *atropine* may help to relieve spasm and irritability of the bowel.

Steroids may be life-saving in acute severe disease; blood and pus disappear from the stools, fever and diarrhoea abate, the patient puts on weight and he feels much better. Prolonged courses of steroids are not given, for it has been found that after such courses the bowel becomes friable and may rupture.

Rectal injections of *hydrocortisone hemisuccinate* are often helpful. The injection is given slowly (over half an hour) last thing at night, when the patient is in bed. An injection may be given nightly for $1\frac{1}{2}$ to 3 weeks.

Surgical Treatment.—When months and months of conscientious medical treatment have failed to cure the patient, and he is still seriously ill, the only hope of cure may lie in giving the colon a complete rest by diverting the fæces from the diseased organ. An opening is made into the lower end of the small intestine (ileum) so that the fæces escape through the incision in the abdominal wall (ileostomy). The semi-fluid faeces pass into a bag which is worn permanently. Ileostomy is a life-saving operation, but only patients with severe and intractable disease will consent to it. Unfortunately

ileostomy must usually be permanent. Colectomy (excision of the colon) is usually performed as well, to remove the diseased part.

Prognosis.—Among those who are ill enough to need hospital treatment the mortality is high—perhaps 30 per cent. die within 5 years of the onset of the illness. Some of the rest recover completely, but many suffer a long drawn out illness with frequent relapses. Many of those who have an ileostomy do very well.

It is all-important to be hopeful and re-assuring when nursing colitis patients. The patient is apt to be appalled if ileostomy is suggested. He should be put in touch with the Ileostomy Club, the members of which have all had the operation. A member will visit the patient for whom the operation has been recommended. The patient is always far more impressed by seeing a healthy-looking person who has had the operation, and who is living a normal, active life, than by anything the doctor can tell him. The ex-patient explains how he manages the ileostomy and how little it interferes with his activities. Such a visit may completely transform the patient's attitude of fear and disgust to one of hope and confidence.

Mr. N., aged 48, a mechanic, had had violent diarrhœa for a fortnight. He had been very worried before that, having just lost his job. On admission to hospital he was passing about twenty stools a day, consisting chiefly of blood and pus. He was given a non-irritating diet, with added vitamins, morphia to control the diarrhœa and several blood transfusions, as he rapidly became anæmic (Hb. 36). In spite of this he got worse and had to have an ileostomy. He was in hospital for about four months and was discharged in good health, having gained over three stone in weight.

(6) Crohn's Disease (Regional Ileitis)

This disease is not very uncommon; it mainly attacks young men. The cause is unknown. Symptoms usually start gradually, though the onset is sometimes abrupt. The patient has attacks of colicky abdominal pain and diarrhœa; the stools may be bloodstained, but are never loaded with blood and pus, as in ulcerative colitis. Fever, anæmia and loss of weight are also common.

Complications.—Intestinal obstruction, perforation of the bowel or fistula formation may occur. (A fistula is an abnormal channel between one organ and a neighbouring one, or between an organ and the surface of the body).

Cause of the Symptoms.—A segment of bowel—usually near the lower end of the small intestine—is swollen, inflamed and ulcerated. Sometimes several neighbouring segments of gut are affected. It can readily be seen how disease of this nature may lead on to intestinal obstruction, perforation or fistula formation.

Treatment.—In the early, acute stages, bed rest is advised. A low-roughage diet is given, as in ulcerative colitis. Anæmia, if present, is treated. Steroids are given if acute symptoms persist in spite of these general measures.

Operation may be necessary if medical treatment fails, or if obstruction, perforation or fistula formation occur.

Prognosis.—The course in regional ileitis is very variable. The disease may clear up completely, but relapses are common. Occasionally the patient goes steadily down hill in spite of treatment.

(7) Abdominal Tuberculosis

This disease is much less common than it used to be. Abdominal tuberculosis is caused by swallowing tubercle bacilli, which may be present in the milk from tuberculous cows. Patients with pulmonary tuberculosis may infect their bowels by swallowing their own sputum. Milk-borne infection is commonest among children and young people; it does not occur in countries where tuberculosis among cows has been stamped out.

Swallowed tubercle bacilli may attack the small intestine, causing tuberculous ulcers there, or they may pass through the bowel wall along the lymphatic vessels to infect the abdominal lymph glands and the peritoneum.

Tuberculosis of the intestines (tuberculous enteritis) causes diarrhœa, abdominal pain, fever, wasting and anæmia. The patient with *tuberculous peritonitis* may have any of these symptoms, but in these cases constipation is commoner than diarrhœa. The abdomen may be swollen with fluid (ascites) or full of irregular, doughy masses, which represent matted folds of peritoneum and coils of intestine. Abscesses may occur in severe cases, and may discharge fæces if both the intestine and abdominal wall are involved.

Treatment consists of prolonged rest in bed, with good nursing, fresh air and nourishing food, as in pulmonary tuberculosis. If there is much diarrhœa the patient should avoid irritating food, as in ulcerative colitis. Anti-tuberculous chemotherapy (see p. 117) will usually arrest or cure the disease in all but the severest cases.

(8) Intestinal Diverticulosis and Diverticulitis

Diverticulosis means the presence of numerous small pouches arising from the wall of the bowel. The lower part of the colon is usually affected. Many healthy elderly people have diverticulosis,

which of itself causes no symptoms. The condition is often discovered by accident during the course of an X-ray examination. No treatment is necessary.

In a small proportion of cases the diverticuli become infected—*diverticulitis*. This causes attacks of pain and tenderness in the left



Fig 43 Diverticulitis

The diverticuli of the descending colon are shown up by little blobs of barium. Normal ascending colon (on left of picture)

lower abdomen. If a diverticulum perforates a pelvic abscess or general peritonitis may follow.

Treatment. During an acute attack the patient should stay in bed and take a fluid diet. Lubricants such as liquid paraffin may be helpful. Between attacks the diet should be non-irritating as in ulcerative colitis. A course of antibiotics (e.g., one of the tetracyclines) is often given. Operation may be necessary in severe cases.

A Note on Diarrhœa and other abnormalities of the Stools

Diarrhœa is a sign of intestinal overaction and hurry. Normally a good deal of water is removed from the bowel contents in the colon; in diarrhœa there is no time for this and a liquid stool is the result.

Diarrhœa is very often a sign of bowel *inflammation*—e.g. in ulcerative colitis (p. 179), typhoid fever (p. 316), dysentery (p. 321), and bacterial food poisoning (p. 321). Diarrhœa may also occur in other diseases of the bowel—e.g. cancer. Often, however, diarrhœa is the result of toxic conditions, such as uræmia (p. 200), in which the bowel itself is not diseased. It accompanies many infections, especially among babies (p. 379). It is a frequent symptom in thyrotoxicosis. Mild diarrhœa is common at times of emotional tension—for instance, during examinations.

The *treatment* of diarrhœa is that of the cause. Kaolin and pectin preparations tend to absorb toxins and may check mild or moderate diarrhœa. Occasionally it is justifiable to give a constipating drug such as opium in exhausting diarrhœa, but this is not usually advisable, for diarrhœa is nature's way of getting rid of poisons in the gut, bacterial or otherwise. In severe diarrhœa the patient becomes depleted of fluid, and may require intravenous infusions.

The nature of the stools. Nurses should note any abnormalities of their patients' stools and report them to the doctor, saving the stool for inspection if necessary.

In simple diarrhœa the stools are liquid and light brown as a rule. In infantile diarrhœa (p. 379) the stools are often green. In ulcerative colitis and severe dysentery the stools contain blood, pus and mucus—signs of severe inflammation of the bowel wall. In typhoid fever the stools may be liquid and greenish—"pea soup" stools.

Other abnormalities of the stools.—In infective hepatitis (p. 174) and obstructive jaundice (p. 179) the stools are pale greyish—"clay-coloured"—because they lack the brown pigments in the bile which give the stools their normal colour, and also because they contain too much fat, which cannot be properly absorbed in the absence of bile.

The stools are pale and bulky in cœliac disease (p. 374) because they contain too much fat, although there is no lack of bile in this condition.

In bleeding peptic ulcer (p. 166) the stools are *black* (melæna) because they contain altered blood. Small quantities of blood need special tests to detect them. Unaltered blood appears in the stools if the bleeding occurs near the lower end of the alimentary tract—e.g. in ulcerative colitis and piles.

Patients who are taking iron preparations have dark stools, but they do not have the tarry appearance of melaena stools.

Constipation (p. 33) and intestinal infestation (p. 348) are discussed elsewhere.

URINARY DISEASES

(1) Pyelitis and Cystitis

INFECTIONS of the urinary tract are extraordinarily common, especially among women, and cases of pyelitis and cystitis will be familiar to all nurses in general wards. Patients may be admitted to hospital for treatment of an acute attack, or for the investigation and treatment of chronic or recurrent infection. Women seem to develop urinary infections when they are kept in bed for whatever cause; this is one reason why patients should not be kept in bed too long.

(a) Acute Pyelitis

The disease generally begins suddenly, with pain and tenderness in the loin, shivering, headache and vomiting; in children there may be convulsions. The patient repeatedly passes small amounts of urine, often with much smarting and pain. The temperature shoots up to 103° or more. Sometimes there are no urinary symptoms, but only those of fever and general ill-health. In every case the diagnosis depends on the examination of the urine.

The Urine in Pyelitis.—In the acute stage the urine is often scanty and highly coloured and it usually contains albumin. The urine looks hazy and, on standing, a thick white sediment is deposited. This sediment tends to form “ropes” on pouring from one glass to another. On examining a drop of urine under the microscope, including some of the deposit, the latter is seen to consist of millions of rounded, speckled *pus cells*. Occasionally *red blood corpuscles* are seen; they look smaller and smoother than the pus cells. In most cases the urine is seen to be swarming with countless *bacteria*, dashing this way and that in what seems to be a frenzy of activity. These bacteria in their millions are the cause of the haziness of the urine already noted.

If the urine contains pus and organisms which are visible under the microscope we can be practically certain the case is one of pyelitis or cystitis. A *catheter specimen* will be required for the more thorough examination which is done in the laboratory, where the pathologist will be able to identify the organism causing the infection, and the drugs to which it is sensitive. A non-catheter specimen is no good

for this purpose, because it is always contaminated by organisms from the rectum or vagina.

Nurses should seize every opportunity of looking at their patients' urine under the microscope. They will find that the cases become much more interesting, and, if there is a ward microscope, there is no reason why they should not learn to use it



Fig. 44 Drawing of a film of urine from a case of Pyelitis, seen under the microscope. Note rounded pus cells and minute rod-shaped bacilli. (The two large cells just above the centre are epithelial cells from the lining of the bladder.)

What can be learnt from the urine described above? It tells us that there is inflammation of some part of the urinary tract, caused by a mobile organism and resulting in the formation of *pus*. The organism causing pyelitis and cystitis is most often *Escherichia coli*, a normal inhabitant of the healthy colon, but many other organisms can infect the urinary tract.

The urine does not tell us which part of the urinary tract is inflamed, but the pain and tenderness in the loin suggest that the outlet of the kidney or *renal pelvis* is involved (pyelitis). The whole series of organs—renal pelvis, ureter, bladder and urethra—through which the urine passes on its way to the exterior, is lined with a

similar membrane, excepting the last part of the urethra. Infection of one part of this tract often leads to infection of another, so that pyelitis and cystitis (inflammation of the bladder) are often found together.

The infection may spread to the interior of the kidney itself, causing *pyelonephritis*. This is a more serious disease which may impair the function of the kidney.

Causes of Pyelitis.—Any urinary tract which is *injured* or *obstructed* is apt to become infected. Thus, kidney or bladder stones are often associated with urinary infection. In *pregnancy* the pressure of the uterus may delay the outflow of urine; pyelitis is very common in pregnant women. *Congenital deformities* are sometimes the cause of urinary obstruction and chronic pyelitis. Urinary infections are common in retention of urine, whatever the cause; thus, enlarged prostate in an old man may cause retention of urine, cystitis and pyelitis. In many cases of urinary infection, however, no cause can be found.

Treatment of Pyelitis.—The patient with acute pyelitis needs the usual treatment of a feverish illness—rest in bed, warmth, and plenty to drink.

If the urine is highly acid, the pain and frequency of micturition may be relieved by large doses of alkalis (e.g. sodium citrate 45 gr. + sodium bicarbonate 45 gr.).

The *urinary infection* can frequently be cured by a course of one of the *sulphonamides* especially if *E. Coli* is the infecting organism. These drugs are excreted in the urine in high concentration, and thus have every opportunity of meeting and helping to destroy the organisms causing the infection. Whichever drug is chosen—perhaps sulphadimidine is the most effective—is given in full doses (see p. 97 for the schedules in common use). The total course consists of 25–30 gm. and lasts for four to seven days. At the end of the course a catheter specimen of urine is sent to the laboratory and will usually prove to be sterile. The patient must drink at least five pints of fluid daily, as in all cases under treatment with the sulphonamides. The excess fluid helps, in any case, to wash away the pus and organisms from the inflamed surface.

If the infection fails to respond to sulphonamide treatment the pathologist will probably be asked to report on the drug sensitivity of the infecting organism. The patient may then be treated with an *antibiotic* to which the organism is sensitive, for instance, chloramphenicol or tetracycline. Some cases of resistant pyelitis respond to treatment with *mandelates*. Mandelates make the urine so strongly

acid that it becomes lethal to organisms. This effect is only possible in a concentrated urine, so, while mandelates are being given, the patient must drink no more than two or three pints of fluid a day. This would be distressing to a patient with high fever, so mandelates are not used in acute pyelitis.

If, in spite of intensive chemotherapy, infection persists, further investigations will be necessary (see below).

Chronic Pyelitis and Pyelonephritis

A patient who has had acute pyelitis often suffers from relapses and the condition may become chronic; in other cases it is chronic from the first. The *symptoms* in chronic cases are often vague—e.g. general ill-health, headache, loss of weight, anæmia. From time to time there may be some pain in the loin or frequency of micturition.

In severe cases the patient gradually goes downhill and develops the symptoms and signs of renal failure (p. 200). Severe hypertension may develop in some cases.

The *urine* is often normal except during relapses; sometimes there is persistent albuminuria. Pus cells can be found from time to time and cultures of the urine may then be positive.

Treatment.—During acute exacerbations treatment with sulphonamides or antibiotics, according to the organism present, is given as in acute pyelitis. *Surgical treatment* may be necessary, if, for example, kidney stones are present. Occasionally only one kidney is diseased, and its removal is curative. In general, however, no treatment is effective in chronic pyelonephritis with renal failure.

(c) Cystitis

Acute cystitis is a feverish illness marked by extreme frequency of painful micturition. There is a constant desire to pass urine but only a few drops may be produced each time. The urine causes much smarting, and there is severe lower abdominal pain, especially just after micturition, when the inflamed walls of the empty bladder come into contact.

The *treatment* of cystitis is like that of pyelitis. Chronic cases may require extensive investigation and, sometimes, surgical treatment.

Cystitis may be caused by careless catheterisation. The most scrupulous asepsis is always necessary when passing a catheter especially if a patient needs catheterising often.

Special Investigations in Cases of Urinary Disease

Urine Culture.—The organism causing the infection can usually be identified, and also the drugs to which it is sensitive.

Blood Urea.—If this is raised it means that kidney function is impaired. (Normal: 15–45 mgm./100 ml.)

A *straight X-ray* of the abdomen may reveal stones in the kidney or bladder.

Intravenous Pyelogram (I.V.P.).—A dye such as diodone is injected intravenously and X-rays are taken at intervals of from 5 to 45 minutes later. Diodone is excreted by the kidney and shows up the kidney pelvis, ureters and bladder in the X-rays. Any distension or distortion of these structures can be noted. A diseased kidney may be unable to form urine or excrete diodone; no shadow will then be seen on that side. An I.V.P. is not done if the blood urea is above 60 mgm./100 ml.

Cystoscopy.—An instrument is passed by means of which the interior of the bladder and the orifices of the ureters can be viewed.

Ureteric Catheterisation.—By means of a cystoscope, a ureteric catheter can be passed up each ureter in turn. In this way specimens of urine from each kidney separately can be obtained. In addition, diodone can be injected into each renal pelvis and X-rays taken (*retrograde pyelogram*).

Renal Biopsy.—Using a special long needle and trocar minute quantities of tissue are withdrawn from the kidney itself and examined microscopically. This may clinch the diagnosis in obscure cases of pyelonephritis, when pus is only occasionally present in the urine.

Renal biopsy can only be carried out in hospitals where staff have been trained in the meticulous technique which is necessary.

(2) Nephritis (Bright's Disease)

(a) Acute Nephritis

The patient is usually a child or young adult who is just recovering from an infection of some kind. Most often this is tonsillitis or scarlet fever—infections caused by hæmolytic streptococci. Other streptococcal infections may be followed by acute nephritis.

The symptoms of acute nephritis usually appear suddenly. *Blood in the urine* is often the first and sometimes the only symptom. The urine may be bright red for a day or two, or, if less blood is passed, it is smokey or brownish in colour. *Oedema* is the other most common symptom. The patient looks puffy about the eyes and face, especially on awakening. Later the feet, legs, genitals and other parts of the body may become swollen.

The patient with bloodstained urine and puffy eyes may feel quite well in himself; sometimes all his symptoms clear up in a few days or weeks. In severe cases, however, there may be fever, severe headache, vomiting, breathlessness, visual disturbances and, finally, delirium and coma. In such a case the *blood pressure* is raised and the urine is scanty (*oliguria*). These very severe cases of acute nephritis may be fatal, but fortunately the great majority of patients recover

—sometimes after a long illness with several relapses. Occasionally the patient with acute nephritis goes on to develop *chronic nephritis* (see below).



Fig. 45 A child with Acute Nephritis, showing oedema of the face

The Urine in Acute Nephritis.—Typically, the urine is *bloodstained*, *albuminous* and *scanty*. As we have seen, blood in the urine is oftenthe first symptom. In other cases, though the urine looks normal, red blood cells can be found with the microscope. In addition, *casts* can be seen under the microscope. These are sausage-shaped structures compacted of blood and debris. The urine invariably contains *albumin*. Finally, the urine may be *scanty*. This is much the more serious sign, as severe and prolonged oliguria is a sign of renal failure (see below). However, many patients with acute nephritis pass normal volumes of urine throughout.

The Blood in Acute Nephritis.—If the urine is scanty, we would expect the blood to contain an excess of substances normally got rid of in the urine. This is just what we do find. The *blood urea* level is raised. As urine is normally on the *acid* side, the blood in these cases may contain an excess of acid and acid salts. If the patient is passing normal volumes of urine, however, his blood may be normal too.



Fig. 46 Urine Film showing casts

The Kidneys in Acute Nephritis.—By examining the kidneys of patients who have died of nephritis we find the clue to many of these symptoms and signs. (Renal biopsy is occasionally done, but is not often justifiable in acute nephritis, as the diagnosis is usually clear.) In acute nephritis the kidney filters—the glomeruli—are inflamed. Glomeruli are minute tufts of capillaries, and their function is to filter off waste substances from the blood. The fluid filtered off by each glomerulus passes down a *tubule* where it is converted into urine. The urine from the million or so glomeruli and tubules in each kidney collects in the renal pelvis, whence it descends to the bladder.

In acute nephritis the glomeruli are swollen and choked with blood and inflammatory exudate. This, of course, slows down their filtering action, so that less and less urine is produced. Urea and other

ingredients of normal urine are dammed back in the blood, and albumin and blood escape from the inflamed glomeruli into the urine. Some of the blood cells which escape from the glomeruli get

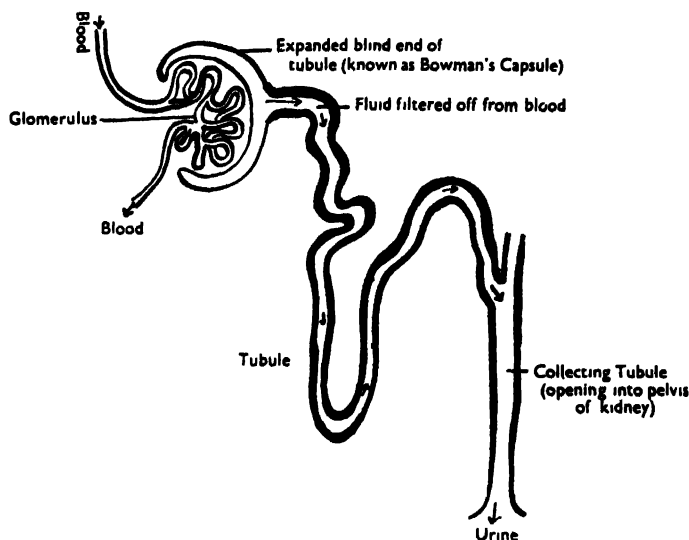


Fig. 47. Diagram of a Kidney Glomerulus and Tubule.

The glomerulus fits closely into the expanded blind end of the tubule so that waste products can easily be filtered off from the blood. The fluid which is filtered off undergoes certain changes as it passes down the tubule, and finally urine is collected up in the kidney pelvis.

packed into the tubules, and, after being moulded into tubular form, appear in the urine as *casts*.

Oedema in nephritis occurs because water accumulates in the body instead of being passed out by the kidneys, and also because capillaries all over the body are damaged, as well as the glomerular capillaries; damaged capillaries leak fluid into the tissues.

Treatment of Acute Nephritis.—*Bed rest* until the acute stage is over is all that is required in mild cases. These patients used to be kept in bed as long as red cells could be found in the urine—often for months; much shorter periods of bed rest are generally advised today. Should the patient relapse on getting up he will have to go back to bed. The patient should remain under medical supervision for a considerable time and a long convalescence may be necessary.

Diet.—This should be *low in salt*, for much salt increases oedema, and, moreover, the failing kidney tends to retain salt. If the blood urea is raised, the diet should be *low in protein*, which, when broken down in the body gives rise to urea. Thus, meat, fish, cheese, eggs, and milk are forbidden or reduced; bread, butter and jam are allowed.

(If the patient is too ill to take solid food glucose (150–200 gm. daily) is added to his allowance of fluid.) If the volume of urine is reduced, the fluid the patient drinks must be reduced too—usually to 500 ml. plus an amount equal to the volume of urine passed during the previous 24 hours.

Drugs.—As a rule drugs are used in the treatment of symptoms only. If signs of infection are present antibiotics are prescribed.

The treatment of severe acute nephritis, with oliguria, is that of acute kidney failure (see below).

In all cases of nephritis the fluid *intake and output charts* must be kept meticulously. The volume of urine passed in 24 hours is the most important single sign in assessing the severity of the illness. Prolonged oliguria is a sign of acute kidney failure—a medical emergency with a serious prognosis, often necessitating difficult and complicated treatment. Diuresis—an increase in the volume of urine passed—is usually the first sign that the patient has turned the corner.

(b) Chronic Nephritis

The early symptoms here are very varied. The onset is insidious. The patient first feels weak, tired, and loses weight; or his ankles swell; or he is breathless; or he suffers from headaches. Sometimes albumin is discovered in his urine on routine testing before any of these symptoms have appeared.

Occasionally a patient who has had acute nephritis appears to recover but later develops chronic nephritis. In such cases albumin and blood cells can usually be found in the urine during the “latent” period when the patient seems quite well.

The patient with chronic nephritis often continues for months or years with only minor symptoms. At length, however, he begins to go downhill—often very rapidly. He feels sick, vomits and is always thirsty. He looks ill, sallow and pale; his skin is dry; his breath smells urinous; his blood pressure is high. Muscle cramps and twitching are common, and so is itching of the skin. Headache is sometimes a torment. The patient is usually breathless, and the respiration may become “hissing” in character, or Cheyne-Stokes respiration may be seen. In the final stages nose bleeds and gastrointestinal hæmorrhages are common. During the last weeks of life the patient often develops pericarditis. Most patients die in coma—sometimes after a series of convulsions; some, however, die of heart failure, pulmonary œdema, infection or cerebral hæmorrhage.

Investigations.—The *urine* is copious, pale, and of a constant specific gravity around 1010. It contains albumin, and often, red

blood cells and casts. The *blood* is often normal in the early stages of the disease; later, severe anæmia is common, and the blood usually contains an excess of urea, potassium, and acid salts, and too little sodium and calcium.

If the kidneys are examined post mortem, they are found to be shrunken and scarred, and, microscopically, there is widespread disorganisation and destruction of both glomeruli and tubules. These appearances will also be found in renal biopsy specimens.

Explanation of the Symptoms.—Chronic nephritis causes *chronic renal failure*, which is more fully described below. Like many other kidney diseases it also causes *hypertension*; the reason for this is not fully understood. Hypertension may cause breathlessness, heart failure, pulmonary œdema, retinitis and cerebral hæmorrhage, as in cases of high blood pressure from other causes; the other symptoms result from renal failure.

Treatment.—Chronic nephritis is always fatal (though often not for years), so treatment must be palliative. There is no point in hedging the patient about with restrictions but he may feel better if his dietary proteins are moderately reduced. The more fluid he drinks the better—dehydration, not œdema, is the danger in chronic nephritis. Symptoms such as headache are treated as they arise. Chlorpromazine may relieve vomiting and distress. Blood transfusions are given for anæmia. Special measures to deal with the metabolic disturbances of renal failure are described below.

(c) Nephrotic Syndrome (Nephrosis)

This syndrome, or group of symptoms, can be caused by a number of quite different diseases—for example, amyloid disease, diabetes, and disseminated lupus. It may also follow acute nephritis, when it is called *subacute nephritis*. In most cases the cause is unknown. *Massive generalised œdema* is the main and often the only symptom in the nephrotic syndrome. The œdema is of gradual onset. First the patient's legs swell, then the genitals and abdomen, and finally the face. The patient sometimes becomes unrecognisable within a few days or weeks. Clinical and X-ray examinations show that he has ascites and bilateral pleural effusions.

The œdema may persist for many weeks, or, after a sudden diuresis it may melt away. Sometimes remissions and relapses alternate for years. The nephrotic patient has little resistance to infection and may die of pneumonia at any time. Sometimes, however, the patient recovers completely. In some cases the blood pressure rises as œdema subsides, and signs of chronic nephritis appear.

Investigations.—The *urine* is heavily loaded with albumin. Blood and casts are usually absent

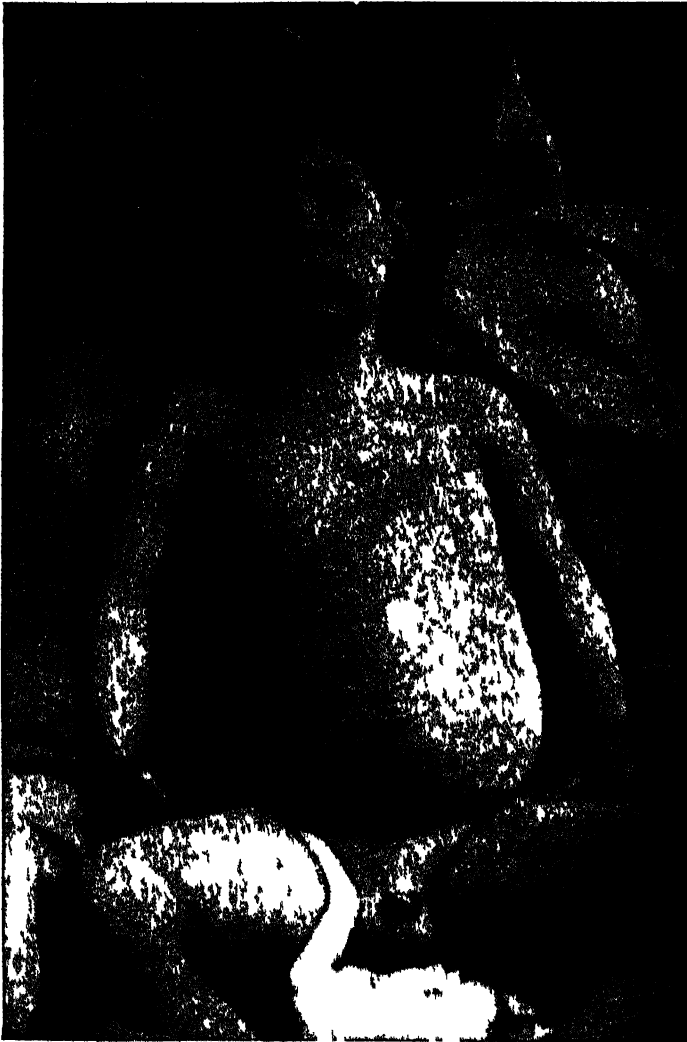


Fig 48 A child with Nephrosis showing generalised oedema

The *blood* contains *too little albumin*—perhaps 1·5 G. per 100 ml. instead of 4–5 G. There is an excess of *cholesterol*—a fat-like substance which may be so abundant that the serum is milky, as was first noted by Bright in 1834. The *blood urea* is usually normal or nearly so.

Explanation.—The œdema is mainly due to the reduced amount of *albumin* in the plasma. Albumin helps to hold fluid in the circulation; the loss of plasma albumin means that fluid escapes into the tissues, resulting in oedema. The plasma albumin is low because albumin is pouring out of the body in the urine. The *cause* of the albuminuria is a disorder of the glomeruli. The cause of the damage to the glomeruli is often obscure.

Treatment.—The patient is usually kept in bed, at least in severe cases. His *diet* should be low in salt and high in protein. *Salt restriction* is necessary because œdema fluid contains salt, and salt in the food helps the body to retain fluid. Plenty of *protein* is required to make up for the protein lost in the albuminous urine.

Steroids often have a dramatic effect in nephrotic cases; diuresis occurs and œdema clears up. Treatment may have to be continued for a long time—sometimes indefinitely. The smallest effective dose is given.

Renal Failure

The patient whose kidneys are failing is always seriously ill and may die. This is because the body's tissues can only live if the composition of the blood plasma and the tissue fluids is kept constant, and the kidneys are the chief regulators of the body fluids. Thus, they keep the balance between acid and alkali, and between water and electrolytes (salts of sodium, potassium, calcium, etc.). This is possible because the kidneys excrete more of a substance which is present in excess and keep back whatever is needed in the body. Thus, if a person drinks copiously, the extra fluid is soon got rid of in the urine, which for some hours will be *copious* and *dilute*. On the other hand, after severe vomiting with a serious loss of chloride, little or no chloride is present in the urine. In addition to these regulatory functions, the kidney gets rid of *urea* and other products of protein breakdown.

When the kidneys can no longer perform these vital tasks the symptoms of renal failure occur.

Acute Renal Failure (Acute Uræmia)

This serious emergency is usually caused by a gross impairment of the blood supply to the kidney. This may occur in severe shock, whatever the cause—burns, other severe injuries, septic abortion, or accidental ante-partum hæmorrhage—and in dehydration, such as may occur after severe diarrhœa and vomiting. In severe shock the blood pressure is very low, and as a result, so little blood may reach

the kidneys that they cannot function normally and *renal failure* occurs—just as *heart failure* occurs if the blood supply to the heart is impaired. Renal biopsy in such cases shows that many of the cells lining the kidney tubules are necrotic (dead) so that the condition is called *tubular necrosis*. (In *cortical necrosis* the damage is so severe that the whole cortex—the outer part of the kidney—is dead: cortical necrosis is always fatal, unlike tubular necrosis from which complete recovery is possible.)

Renal failure can also occur if the kidney is poisoned by *bacterial toxins* or *chemical poisons*, but this is less common. As we have seen, it occurs, though rarely, in acute nephritis. Other severe infections may be complicated by renal failure. *Chemical poisons* include mercury compounds, carbon tetrachloride, and also certain drugs—e.g. sulphonamides (p. 97). *Incompatible blood transfusion* can cause renal failure.

Signs and Symptoms of Acute Renal Failure.—The first symptom is *oliguria*—the passage of very little urine. (Complete suppression of urine—*anuria*—is very rare.) The 24-hour output of urine is often only a few ounces; the urine is usually dark in colour, contains albumin and the specific gravity is constantly around 1010.

At this stage there are few symptoms, apart from those of the disease causing the renal failure. After some days the patient's condition deteriorates; vomiting, weakness, hiccup, muscular twitching, confusion, stupor, convulsions, and finally coma occur in severe cases. Without special treatment the patient who is oliguric for more than a fortnight is likely to die in coma.

The first sign of recovery—occurring naturally or as a result of treatment—is *diuresis*—the passage of increased volumes of urine. The patient's clinical condition does not at first improve—it may even deteriorate. But after the daily output of urine has been above a litre or two for some days, improvement is rapid in uncomplicated cases, though the patient is usually weak and emaciated and will require a long convalescence. He often has a voracious appetite and this helps him to regain lost weight and strength.

Investigations.—Throughout the *oliguric stage* the blood urea rises steadily—perhaps to 500 mgm. per 100 ml. or more. The plasma potassium also rises and the plasma bicarbonate *falls*—a sign of acidosis. If the plasma potassium is above 7 or 8 mEq. per litre (normal 4–5 mEq.) the electro cardiogram shows characteristic changes.

Treatment.—The treatment of this grave emergency calls for medical and nursing skill of a high order. The aim of treatment is to tide

the patient over until his kidneys start working again. Little or nothing can be done to hasten the recovery of the kidney itself.

Nothing must be given to the patient—by mouth or intravenously—which the kidney cannot excrete—no excess fluid, no salts of any kind, no fruit or fruit juices (they contain potassium), no protein foods (urea results from protein breakdown).

Both fluid and food is supplied as 20 per cent. glucose solution of which the patient has a daily allowance of 500 ml. plus an amount equal to the volume of urine passed during the previous 24 hours (500 ml. is about the amount of fluid lost daily in sweat, breath and fæces). This quantity of fluid avoids both dehydration and water logging, and the glucose supplies energy. The patient drinks the solution if he can. If he is comatose, the solution is given by gastric drip. If vomiting makes this impossible, the fluid is given by infusion into the *superior* or *inferior vena cava*; a cardiac catheter is introduced into a vein in the arm or leg and passed into the corresponding vena cava. This is necessary because concentrated glucose solutions cause thrombosis if given into small veins.

Although these measures may take care of fluid and electrolyte balance, they do nothing to get rid of waste products such as urea. These waste products go on accumulating even though no protein food is eaten, because a certain amount of breakdown of the body protein is going on all the time, and this releases urea and other waste products, and also potassium, into the circulation.

To help to get rid of excess potassium *cation exchange resins* can be used. These substances are given by mouth; they take up potassium and release sodium, thus lowering the blood potassium. Injections of *insulin* also help to lower the blood potassium; the reason for this is obscure.

If the patient still gets worse—if stupor deepens into coma, while the blood urea and blood potassium continue to rise—*hæmodialysis* (treatment with an artificial kidney) may be advised. The patient's blood circulates through an apparatus in which it is separated from sterile normal saline by a thin membrane; urea and other waste products diffuse through this membrane, which keeps back protein, glucose, etc. Only a few hospitals are equipped with an artificial kidney and patients often have to be transferred by road or air to reach them.

The oliguric stage may last for a few days to a fortnight or more. When *diuresis* occurs the patient is not yet out of danger; for the body may all at once lose *too much potassium* and other electrolytes (causing weakness). Vigilance must be maintained; the fluid balance charts

and blood tests will show when extra fluid or extra electrolytes are required. After a few days, if all goes well, the blood urea starts to fall; the patient's diet can then be gradually increased.

Other measures.—If the patient is anæmic he may be given a transfusion of *packed red cells* (to avoid giving excess fluid). If the blood sugar rises excessively, insulin is given. Antibiotics are usually given to guard against infection. (N.B.—Streptomycin is *never* given in renal failure, as the drug is not excreted and accumulates in the blood, causing toxic effects such as giddiness or deafness.)

Results of Treatment.—These depend on the *cause of the uræmia* and on the *skill and resources* of the medical and nursing team. The outlook is fairly good in many medical and obstetrical conditions; it is apt to be grave in surgical emergencies, such as crushing injuries and burns. If recovery occurs *it is usually complete*, for the kidney has suffered no irreparable damage in these cases.

Chronic Renal Failure (Chronic Uræmia)

This is usually the result of chronic renal disease (such as chronic nephritis, chronic pyelonephritis, hypertensive kidney disease, or congenital abnormalities such as polycystic kidney). Most of these diseases are incurable and so is the renal failure they cause. Chronic pyelonephritis, however, can sometimes be cured, so, as we have seen, it is important—and often difficult—to distinguish this disease from chronic nephritis.

Chronic renal failure sometimes results from urinary obstruction—due, for instance, to enlarged prostate or stones blocking both ureters. These conditions can often be relieved by operation.

Symptoms come on gradually and are very varied. Weakness, loss of appetite, loss of weight and anæmia and shortness of breath are common. Nausea, vomiting, diarrhœa and stomatitis; extreme dyspnœa with hissing respiration; copious urine and dehydration; foul “urinous breath”; slow irregular pulse; muscular twitching, tetany, convulsions and coma—any or all of these may occur in chronic uræmia. Severe anæmia is usually present in the late stage. Pericarditis may occur towards the end.

Investigations.—The *urine* is copious and of a constant specific gravity of 1010. It usually contains albumin. The *blood* contains an excess of urea, potassium and acid salts, and too little sodium and bicarbonate, and, often, too little calcium.

Explanation of the Symptoms.—The failure of the kidney's regulating and excreting functions explains some of these symptoms, while others are due to the “toxic” effects of the disease. Thus, *hissing*

respiration is caused by acidosis, *cardiac irregularities* by high blood potassium, and *muscle twitching* by a low blood calcium. The kidney is unable to adapt the urine to the body's needs, so that the specific gravity remains constantly at 1010, whatever the patient eats or drinks. Large volumes of this dilute urine are passed (unlike what is seen in acute renal failure). This helps to compensate for the kidney's inability to produce a concentrated urine, but it also leads to *dehydration*.

Anæmia, nausea, vomiting, diarrhœa, pericarditis and coma are signs of the general "toxic" state. *Convulsions* may be caused by uræmia or by the high blood pressure which is so often present.

Treatment.—Since most cases of chronic renal failure are due to incurable disease treatment must usually be palliative. The patient may eat and drink what he likes. He is apt to be dehydrated so he is encouraged to drink as much as he can. If vomiting makes this impossible, he may be given an intravenous solution of *sodium lactate*—this relieves both dehydration and acidosis and often improves the general condition markedly. If there is tetany and the blood calcium is low, calcium is given, by mouth or intravenously.

For severe anæmia, blood transfusion, for infection, antibiotics, for headache, pain relievers, and for vomiting, chlorpromazine are given as required.

Prognosis.—Although the diseases causing chronic renal failure are nearly always fatal, the patient often has months or years of reasonable health before symptoms become severe.

DISEASES OF JOINTS AND MUSCLES

THE diseases so far described in this book include many severe and some lethal illnesses. But these dangerous illnesses only account for a small part of the nation's ill-health. In this section we shall study a group of diseases which rarely kill but often cripple, and which account for more loss of working time, suffering and disablement than any other type of illness. National Health Insurance figures show that one-sixth of all sickness among insured workers is caused by "rheumatism," and it has been calculated that the average general practitioner spends about one-tenth of his working day during the winter months attending rheumatic cases.

"Rheumatism" to the layman may mean almost any pain in the back, limbs or joints. Here are some of the many diseases which may cause such symptoms. *Acute rheumatism* has already been described (p. 57).

(1) Rheumatoid Arthritis

The end results of this disease are to be seen in any women's ward or, indeed, in any bus, shop or restaurant. Rheumatoid arthritis is very common and causes a great deal of crippling, both mild and severe. The commonest signs of old rheumatoid disease are the crippled hands with swollen joints and fingers bent away from the thumb, so often seen among elderly women. More severe forms of this disease cause the most dreadful crippling and invalidism, which is especially tragic as young adults are quite often affected, and in them the disease tends to assume its most virulent and disabling forms.

Rheumatoid arthritis is a generalised disease of unknown causation which makes the patient ill as well as crippled. Women are affected about five times as often as men, chiefly between the ages of 20 and 40. The disease is thought to be precipitated by worry, infection or injury in some cases. In others there is a family history of rheumatoid arthritis.

Symptoms.—Before there are any signs of joint disease the patient may feel out of sorts for several weeks. She feels tired, loses weight and sweats profusely; she may become anæmic and her periods irregular. She then notices that certain joints are becoming

stiff and painful. Almost any joint in the body may be affected, but those of the fingers, hands and knees are most commonly attacked. The painful, stiff joints eventually become swollen and tender. The disease affects one joint after another, until in severe cases the patient is extensively crippled.

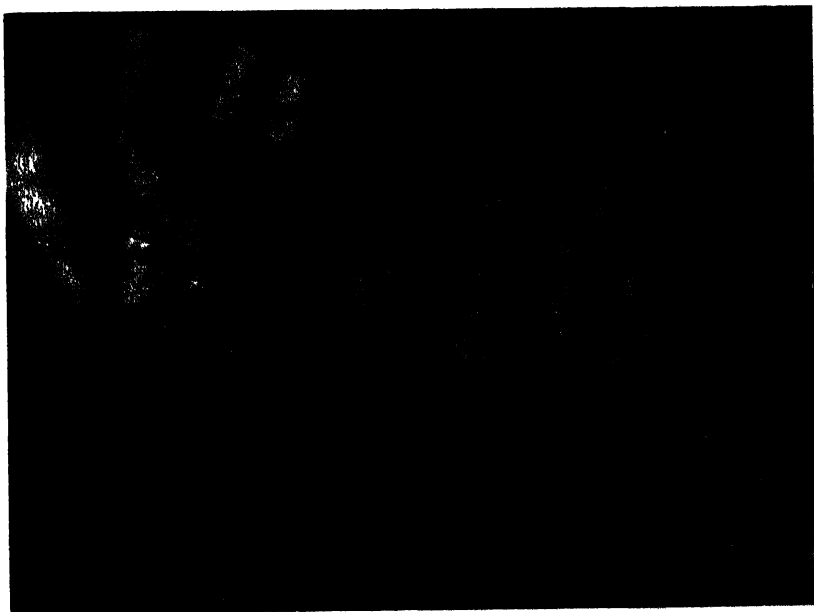


Fig. 49. Rheumatoid Arthritis.

Hands, showing typical deformities (Dr Richard Asher's case)

The disease commonly takes months or years to develop, with frequent though temporary remissions. In some cases the patient may be completely disabled within a few weeks. The younger the patient the more acute the disease; in these acute cases the arthritis is often accompanied by fever. Anæmia is very common and is apt to be severe.

The muscles controlling the affected joints rapidly waste, and the overlying skin is thin and sweaty. The wasting often affects one group of muscles more than another, and the parts are pulled out of position by the overaction of the stronger muscles. In the *hands* this deformity drags the fingers away from the thumb, and the *knees* and *elbows* cannot be fully straightened.

The disease eventually "burns itself out", and the patient may be left with any degree of disability, from mild to crippling. In the

severest cases there are gross deformities, with fixed, immovable joints and severe wasting of muscles.

Investigations.—*X-rays* show that the bones next to the arthritic joints are more porous and less solid-looking than normal bones—*osteoporosis*. The *blood sedimentation rate* is always raised in the active phases of the disease, sometimes grossly so—up to 80 mm. in one hour or more. *Blood counts* show varying degrees of anæmia. The *sheep cell agglutination test* is positive in the majority of cases.



Fig 50. Rheumatoid Arthritis

X-ray of hands shown in Fig 44 Note displacement of bones, rarefaction, and bony union (ankylosis) of some of the joints

The Cause of the Symptoms.—In rheumatoid arthritis the *soft tissues* of the joint are affected first, and the membrane lining the joint—the *synovial membrane*—is inflamed and thickened and finally encroaches on and eats away the actual joint surfaces. Later these damaged surfaces tend to become fixed together (ankylosis) by fibrous or even by bony tissue. The muscles waste, partly because they are not used and perhaps, too, because they are affected by the same toxic process as are the joints. Deformity results when some of the muscles acting on a joint are stronger than others, and pull it out of position. The anæmia, loss of weight, sweating, and the high B.S.R. are all signs that the whole body and not merely the joints is affected by the inflammatory process.

Treatment.—This has largely been by trial and error, for we do not know how to cure rheumatoid arthritis any more than we know what causes it.

In a severe case the patient should be kept in bed in the acute stage and should eat nourishing, appetising food. No special diet is necessary, unless the patient is overweight, when she should go on a reducing diet. These patients are apt to develop bedsores, which must be prevented in the usual way.

The *joints* should be *rested* during the acute stage and *exercised* thereafter. While rest is necessary for the recovery of inflamed,



Fig 51 X-ray of normal hands.

tender, swollen joints, deformities must at all costs be prevented by means of well-arranged pillows, sandbags or even splints. As soon as the acute stage is past the patient must perseveringly move the joints as much as she can, to prevent fixation, deformity and muscular wasting. Massage and special exercises will be necessary in the later stages. The patient should be got up and about at the earliest possible moment, for otherwise there is a great danger of her becoming permanently bedridden and immobile. It needs great strength of mind for the patient to go on exercising her joints, in spite of pain and muscular weakness.

Spa treatment can be valuable, offering as it does an intensive programme of physiotherapy and rehabilitation. Moderately disabled patients can often be helped in running their homes by the use of special gadgets and domestic appliances.

Rheumatoid arthritis patients, as can well be imagined, are apt to be depressed. It is of the utmost importance that doctors, nurses and physiotherapists should inspire them with a spirit of determination and hopefulness. It is surprising what can be achieved even by those who are severely crippled. (See Fig. 52). Renoir continued to paint masterpieces when he could no longer walk and the brush had to be strapped to his hand.

Drugs.—Aspirin is given to relieve pain; it may also have some effect on the rheumatic process. The dose should be the largest the patient can tolerate—e.g. three or four tablets (15–20 gr.) four times a day. *Calcium aspirin* is pleasanter to take than ordinary aspirin and causes less digestive upset.

Another effective pain reliever is *phenyl butazone* (butazolidine) but it often causes toxic reactions, such as blood disorders, which have occasionally been fatal.

Curiously enough, *anti-malarial drugs* such as chloroquine, seem to have a beneficial effect in certain cases. They may, however, cause nausea or vomiting.

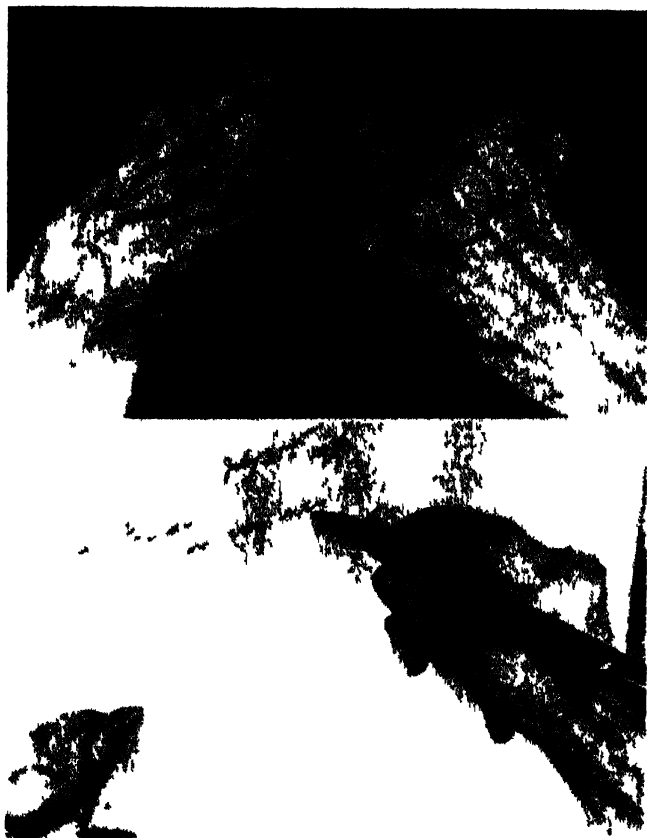
Gold salts appear to bring about a remission in some cases, but they, too, are apt to be toxic, causing dermatitis, nephritis or blood disorders in a considerable proportion of patients. Gold salts are given by intramuscular injection at weekly intervals, starting with a very small dose. The urine is tested for albumin before each dose. If albuminuria or a rash develop the course is stopped.

Steroids.—The dramatic effect of cortisone in rheumatoid arthritis was discovered by Dr. Hench and his colleagues in the U.S.A. in 1950. Pain, stiffness and swelling of the joints are relieved, fever abates, the patient's appetite improves and her spirits rise. As soon as the drug is stopped (and this has to be done slowly), there is a relapse, and the patient is soon as bad as ever. It is often impossible to continue steroid treatment indefinitely, either because it becomes ineffective, or because it has undesirable side effects (p. 60). One of the most serious of these is *osteoporosis*; as we have seen, the disease itself causes osteoporosis and this may be increased by steroid treatment until pathological fractures occur. (*Crush fractures of vertebrae* are not uncommon, causing back pain.)

Injections of hydrocortisone or prednisolone into the affected joints often give temporary relief, and do not have the serious side effects of oral steroid treatment.

Iron should be given if the patient is anæmic, though the anæmia in rheumatoid arthritis is apt to be very resistant.

Surgical Treatment.—Operation to correct deformities may be



July 21st 1950
 My dear friends
 I am very much

Rheumatoid Arthritis

Fig 52 To show how useful arthritic hands can be (a) Grossly deformed hands, (b) Same hands writing, (c) The writing—much more legible than the average doctor's (Dr Richard Asher's case)

necessary in severe cases. Arthrodesis (fixation of joints) gives stability and relief of pain at the cost of permanent stiffness. Arthroplasty—the fashioning of new joint surfaces—is sometimes possible.

Prognosis.—Only a minority of patients—perhaps 15 per cent.—recover completely. The rest are left with more or less disability; a few are completely crippled. With energetic treatment the great majority retain a useful degree of activity.

It is often amazing to see how active and independent a really determined patient can be, in spite of what appear to be grossly incapacitating deformities.

Case History.—Mrs. C., aged 38. The disease started six years ago with pains in the right elbow, and it spread rapidly to all the joints of all four limbs. She has been treated at different times with vaccines, radiant heat and massage, brine baths (at a spa), and three courses of gold treatment. The last course had to be stopped as she developed albuminuria. She is now deformed, crippled and anæmic (Hb. 48 per cent.) and looks years older than her age. She hobbles round the house and does her own cooking, though her husband has to do the washing and heavy cleaning.

Other Varieties of Polyarthritis (Arthritis of Many Joints)

Still's Disease.—This is a form of rheumatoid arthritis affecting children. Enlargement of the lymph glands and spleen is seen, as well as polyarthritis. The arthritis is often very severe and crippling in its effects.

Reiter's Disease.—Here polyarthritis is associated with *urethritis* (inflammation of the urethra). The patient suffers from various micturition symptoms, and, in addition, his *eyes* may be affected too (conjunctivitis or iritis). Another curious symptom is a great thickening of the skin of the soles of the feet. **Treatment** is with salicylates and steroids.

Disseminated Lupus Erythematosus.—This disease may be described here, although arthritis is only one of many possible manifestations. Should the disease affect the joints, the symptoms are similar to those of rheumatoid arthritis, though the patient is usually iller and may be anæmic; the spleen is often enlarged.

In other cases the patient with disseminated lupus erythematosus is admitted with a pyrexia of unknown origin ("P.U.O."), or with heart failure, pericarditis, nephrotic syndrome, purpura, mental symptoms, convulsions or a rash. The rash sometimes starts as a chronic scaly patch on the face (chronic lupus erythematosus). Later the rash becomes more acute, resembling dermatitis, and spreads over the rest of the body.

One or more of these groups of symptoms may occur in any one patient. The disease may last for years, with frequent exacerbations and relapses.

These very varied symptoms are possible because the disease is a disorder of the connective tissue, and so may affect any organ in the body. Disseminated lupus erythematosus is one of a group of *collagen diseases*—collagen being the connective tissue substance which is disordered. Rheumatoid arthritis is sometimes classed as a collagen disease.

Investigations.—Abnormal leucocytes called L.E. cells (lupus erythematosus cells) are seen in stained blood films; this test clinches the diagnosis.

The sedimentation rate is high, the white count and platelet count are often low.

Treatment.—*Steroids* control symptoms in many cases; in some cases they are necessary only during exacerbations, in others, small doses are given indefinitely. For some unknown reason *anti-malarial drugs*, such as chloroquine, are often effective too. They are widely used in the treatment of chronic localised lupus erythematosus of the skin.

Sjögren's Disease.—Some people think this rare disease is a form of disseminated lupus erythematosus. The patient usually has arthritis of the rheumatoid type, and also swellings of the salivary and lachrymal (tear) glands. Saliva and tears are scanty; as a result, the mouth is unpleasantly dry and the eyes inflamed and painful. *Steroids* may be helpful. *Symptomatic treatment* includes the use of eyedrops, and sucking lemons to increase the flow of saliva.

(2) Ankylosing Spondylitis

This is a form of arthritis of the spine; some consider that it is a special variety of rheumatoid arthritis. In the usual form of rheumatoid arthritis the spine is rarely affected, and the disease attacks women much more often than men. Ankylosing spondylitis is much commoner among men. The first symptom is usually pain or stiffness of the lower back. This gets worse, and extends up the spine. In severe cases the whole spine eventually becomes completely ankylosed and the shoulders and hips may be affected too.

Treatment. Rest in bed, with one pillow and boards under the mattress, is necessary in the acute stages. Exercises (including breathing exercises) are most important. The effects of cortisone are the same as in rheumatoid arthritis. *Deep X-ray* therapy may be most effective, but carries serious risks, the gravest being *leukæmia* (p. 156) which develops in a small proportion of cases.

In advanced cases orthopædic treatment may be necessary.

(3) Acute Infective Arthritis

There are several infectious diseases in which acute arthritis may occur. The inflammation of the joints and surrounding tissues is caused by bacteria which have been carried by the blood from the original site of infection. Arthritis is fairly common in gonorrhœa, in certain forms of dysentery and in cerebrospinal meningitis; it is also a rare complication of pneumonia. It is common in the aged, in whom the cause is often obscure and symptoms few or none. The arthritis may be of the rheumatoid type, or it may be limited to one or two joints. The *treatment* is that of the primary disease; the joints

should be treated as in rheumatoid arthritis. The joints are much more likely to recover completely in these cases than in rheumatoid arthritis.

(4) Acroparæsthesia

This common condition mainly affects middle aged women. The patient is usually awakened in the small hours of the morning by tingling and pain in the hand and forearm. Symptoms may also occur during the day. Various causes have been suggested—for instance, pressure on the nerves to the arm by the first rib. At present, however, it is generally thought that the symptoms are the result of compression of the median nerve—the nerve which supplies part of the skin of the hand, and also some of the muscles of the hand and forearm. This nerve passes into the palm of the hand through the *carpal tunnel*. (The carpal tunnel lies in front of the wrist joint and is roofed in by the strong carpal ligament.) The carpal tunnel also contains tendons. Should any of the tissues in the tunnel swell, the median nerve will be compressed.

Treatment. -Steroids may help to reduce swelling and so relieve symptoms. Night splints are often very effective. If symptomatic treatment fails the carpal ligament may be cut at operation; relief of pressure and, in most cases, relief of symptoms follows.

(5) Osteoporosis and Osteomalacia

These two disorders of bone usually occur as complications of other diseases. In both, bones become weak as a result of alteration in their structure.

Bone consists of a protein frame work or *matrix* in which calcium phosphate is deposited. In osteoporosis, the protein matrix is affected; in osteomalacia, the calcium salts are deficient.

Osteoporosis occurs in old age, in various endocrine disorders (e.g. Cushing's syndrome) and—of great importance—after prolonged steroid treatment. In all these diseases the bones are affected because the body's metabolism is disturbed. Osteoporosis also occurs in prolonged immobilisation—thus, the bones of a limb paralysed by poliomyelitis or immobilised after a fracture are often affected.

Osteoporosis is often symptomless, and is then diagnosed only on X-ray, the affected part appearing more translucent or thinner than usual. Often, however, the patient suffers from *backache* if the vertebræ are affected. In senile osteoporosis vertebræ sometimes

collapse, causing spinal curvature and reduction in height. Spontaneous fractures of other bones may occur.

Treatment is that of the cause. Male and/or female sex hormones are sometimes valuable in senile cases. When osteoporosis results from steroid therapy, the latter may have to be discontinued.

Osteomalacia is much less common. It is the adult form of rickets. *Pains in the bones* and deformities are much commoner than in osteoporosis; spontaneous fractures may also occur. *Blood tests* show that the *blood calcium* and *blood phosphorus* are low.

In osteomalacia the bones are short of calcium phosphorus because the diet lacks calcium and vitamin D, or because these substances are not absorbed from the gut, or because too much calcium and phosphorus are excreted in the urine. Osteomalacia is practically never due to a *poor diet* in Britain, though it occurs in India and other countries. Poor absorption of calcium accounts for osteomalacia complicating *steatorrhœa* (p. 148). Osteomalacia may also occur in *kidney disease*, because of excessive loss of calcium and phosphorus in the urine.

Treatment is that of the primary cause, plus large doses of calcium and vitamin D when necessary.

(6) Osteoarthritis

This is a much less acute and less crippling disease than rheumatoid arthritis, chiefly attacking elderly people. Osteoarthritis tends to single out one of the larger joints for attack; the knees, hips and spine are often affected. Any joint which is subject to repeated strain or injury is a candidate for the disease. Thus, osteoarthritis of the hip often follows a badly united fracture of the neck of the femur, while coal heavers are apt to develop osteoarthritis of the spine. Obese women often develop osteoarthritis of the knees about the time of the menopause.

The disease may cause great pain and limitation of movement, but the extensive crippling typical of rheumatoid arthritis does not occur, nor is the patient's general health affected. In osteoarthritis the joint surfaces are damaged, and their covering of glistening white cartilage (whose normal appearance will be familiar to anyone who has prepared meat in the kitchen) becomes ridged and wears thin, until the underlying bone is exposed. The pain and creaking are caused by rough surface grating over rough surface.

Treatment.—No curative treatment is known. Pain relievers such as aspirin may be taken regularly. Overstraining the affected joint

must be avoided, but moderate exercise is valuable. The importance of weight reduction, where an obese patient has osteoarthritis of the knees, hips or spine, cannot be over-stressed. Not only is the extra thirty or forty pounds weight a cruel burden on the damaged joints, but the posture is usually bad in fat people, putting an unnatural



Fig 53 Osteoarthritis of Hip
Note irregularity of the head of the right femur, compared with the smooth, rounded outline on the left, and diminished joint space (The patient was an old lady who had fallen from the loft and injured her hip some years before. No fracture was ever demonstrated) (Dr Richard Asher's case)

strain on the weight-bearing joints, and uneven pressure on joint surfaces. Appetite reducers such as dextroamphetamine sulphate are often prescribed to help obese patients to stick to a rigid reducing diet. Loss of weight is often rewarded by a gratifying loss of symptoms.

† Physical treatment is often valuable, including massage, radiant heat and special exercises to correct any errors of posture which may make the condition worse.

Occasionally surgery is necessary, as in osteoarthritis of the hip, in which fixation (arthrodesis) of the joint is sometimes advised. It may be less disabling for the patient to have a stiff hip than a constantly painful one. Or new joint surfaces may be fashioned out of plastic (arthroplasty).

Treatment

(a) *Treatment of acute attack.*—The patient is put to bed and the affected joint rested completely, if necessary by splinting. If the lower limb is affected the bedclothes are supported on a cradle. *Colchicine* has a dramatic effect in acute gout; in the form of tablets, tincture or mixture, it is given every two or three hours for 12 to 24 hours, until nausea or vomiting occur. The drug is then stopped. If necessary, it can be started again after two or three days.

Steroid treatment is also valuable in many cases of acute gout.

(b) *Treatment of chronic gout.*—The patient should avoid foods rich in uric acid-producing substances, such as liver, kidney, heart, herring, herring roe and sweetbreads. He should also be teetotal. Drugs which help to clear uric acid from the blood may be taken regularly; *probenecid* is effective and so is *aspirin*, as long as a large enough dose is taken (up to 15 tablets a day). During the first few weeks of treatment probenecid may precipitate an acute attack of gout. This is because the drug mobilises uric acid from various deposits, so that the blood uric acid rises. The excess is eventually got rid of in the urine.

The patient often learns by bitter experience what is likely to bring on an acute attack—overeating, drinking, violent exercise, or exposure to cold and damp—and to avoid such activities.

ENDOCRINE DISORDERS

WE come now to the diseases of the *endocrine glands*. The secretions of these glands—the *hormones*—are the body's chemical messengers (the word "hormone" comes from the Greek, meaning "I rouse to activity"). Hormones control or influence an enormous variety of activities—mental and physical growth, sexual development, blood formation, the circulation, the development of bone and many of the chemical processes necessary to the life of every living tissue. Hormones are closely bound up with the emotions. For instance, the palpitations, rapid pulse, dry mouth and lack of appetite familiar to all examination candidates are due to the secretion of *adrenaline*, the hormone responsible for many of the physical symptoms of fear and excitement.

The endocrine system includes the pituitary, thyroid, parathyroid, adrenal and sex glands (ovaries and testes); certain cells in the pancreas produce the hormone *insulin* and other organs may also have some endocrine activity. Some endocrine glands produce several hormones. When a gland is disordered it may produce either *too much* or *too little* hormone; the excess or lack of hormone may result in one of the diseases now to be described.

(1) Diseases of the Thyroid Gland

(a) Thyrotoxicosis

The patient may be a thin, nervy young girl with staring eyes, or a breathless middle-aged woman. Any patient may have any or all of the following symptoms, but younger patients are more likely to suffer from *loss of weight* and *nervous symptoms*, whereas the *heart* is more often affected in older women. Symptoms may come on rapidly or very gradually.

Symptoms and Signs of Thyrotoxicosis

(1) *Goitre*.—The thyroid may be slightly or grossly enlarged, smooth or nodular, or there may be no detectable enlargement. Sometimes an obvious goitre has been present for many years before thyrotoxic symptoms appear.

(2) *Exophthalmos* (protruding eyes).—This is especially common in young women; all degrees of severity are seen from slightly staring eyes to such gross protrusion that the eyes cannot be closed.

(3) *Loss of Weight.*—This is often severe, although the appetite remains good. The patient eats ravenously yet grows thinner.

(4) *Nervous and Mental Symptoms.*—The thyrotoxic patient is usually excitable, emotional and unstable. She blushes easily and



Fig 55 Thyrotoxicosis
Exophthalmic goitre in a young man (Dr. Richard Asher's case)

bursts readily into tears. There is a fine tremor of the fingers when she stretches out her hands. Mild thyrotoxicosis is sometimes difficult to distinguish from anxiety state (p. 402).

(5) *Cardiac Symptoms*.—Palpitations and breathlessness are common. The pulse is rapid (often 100–120) and is often irregular, especially in older patients. Auricular fibrillation is sometimes the first or only sign of thyrotoxicosis in a middle-aged woman. Auricular fibrillation in thyrotoxicosis is very difficult to control with digitalis, and this may give the clue to the diagnosis when there are few other signs of thyroid disorder. If, in an attempt to control the heart rate the dosage of digitalis is increased, signs of digitalis intoxication are very apt to occur.

(6) *Other Symptoms*.—Thyrotoxic patients have warm, moist skins and dislike hot weather. Diarrhoea is common. The menstrual periods are often irregular or cease altogether.

Thyrotoxic Crisis.—Fortunately this is now a rare occurrence. In severe cases of thyrotoxicosis a crisis may be precipitated by an acute infection. An acute exacerbation of symptoms occurs, with fever, uncontrolled auricular fibrillation and mental symptoms, sometimes going on to heart failure or insanity.

Investigations.—These are usually less important than clinical findings.

The *Basal Metabolic Rate* (B.M.R.) is the rate at which the body is consuming its fuel at rest. The B.M.R. is estimated by measuring the patient's oxygen consumption first thing in the morning. It is expressed as a percentage above or below normal for one of her age and size. In thyrotoxicosis the B.M.R. is usually 20–70 per cent. above normal (+ 20 to + 70).

The *radio-iodine uptake* is a test which shows how quickly iodine disappears from the blood and is taken up by the thyroid gland. In thyrotoxicosis the thyroid gland rapidly takes up iodine from the blood.

The Nature of Thyrotoxicosis

The patient with thyrotoxicosis has an overactive gland, which pours out excessive quantities of its hormone (thyroxine). Thyroxine increases the rate of metabolism—the rate of the body's energy consumption. Thus, however much she eats, the thyrotoxic patient loses weight, because fuel is so rapidly consumed in the tissues.

The *rapid pulse* is due to a direct action of thyroxine on the heart muscle, the excitability and other mental changes to its action on the nervous system.

Because thyroxine contains iodine, and because the gland is producing thyroxine so abundantly, the gland avidly takes up iodine from

the blood after a dose of an iodine compound—hence the high radio-iodine uptake.

Treatment.—The over-production of thyroxine may be checked by removing most of the gland at operation (*thyroidectomy*) or by damping down its activity, either with *drugs* or with a *radio-active iodine* compound.

Drug treatment is suitable in many cases. It aims to tide the patient over until a natural remission occurs. A course of drugs usually lasts for several months; large doses are given at first, until the overactive gland has been brought under control when a small, maintenance dose can be given.

Drug treatment usually relieves thyrotoxic symptoms such as loss of weight, tachycardia and nervousness. It does not affect exophthalmos (this may become either better or worse), nor is the goitre reduced in size—it may enlarge under drug treatment. Relapses may occur after drug treatment is stopped; another course may then be given, or the patient treated surgically or with radio-iodine. If too large doses of anti-thyroid drugs are given, myxædema results, but this clears up when the dosage is reduced. The *disadvantages* of drug treatment are the *length* of treatment, the enlargement of the goitre and the possibility of toxic reactions (see below).

Anti-thyroid drugs include the following:

- (a) *Thiouracil* compounds—e.g. methyl thiouracil. The starting dose is about 200–300 mgm. daily, and the maintenance dose is 25–50 mgm.
- (b) *Carbimazole* (“Neo-mercazone”) and allied drugs. The starting dose is about 30 mgm. daily and the maintenance dose about 5 mgm.
- (c) *Potassium chlorate* has recently been used and may prove a useful addition to the anti-thyroid group of drugs.

Drugs of the thiouracil and carbimazole series both cause toxic symptoms in a fair proportion of cases, and patients should be under regular medical supervision. Toxic reactions are most common during the early weeks of treatment; they include fever, rashes, and agranulocytosis. The last is by far the most serious. The white count is usually checked regularly, for the first weeks, but this is not enough, for the white count may fall disastrously in a matter of days or even hours. Therefore the patient must always be warned to report to her doctor at the first sign of *fever, sore throat, or sore mouth*—common early symptoms of agranulocytosis. If the blood disorder is detected in its early stages, it will usually respond to stopping the drug and giving penicillin (p. 154).

Radio-active Iodine.—It has long been known that X-rays depress the activity of the thyroid gland. Radio-active iodine has the same effect, but the irradiation takes place within the actual tissues of the gland. The patient drinks a solution of radio-active potassium iodide. Within a short time nearly all the iodine has been taken up by the thyroid gland. (Most of the iodine in the body is normally stored in this little organ and, as we have seen, in thyrotoxicosis the gland takes up iodine more avidly than normally.) The radio-activity of the iodine only lasts for a week or two, but this is long enough to reduce the over-activity of the gland. If the dose has been rightly estimated—and this is very difficult to do—only one dose is necessary, which makes this form of treatment the simplest of all from the patient's point of view. In successful cases all thyrotoxic symptoms are relieved, except exophthalmos. (Large goitres do not disappear.) The full effect of the treatment is not apparent for about three months. If the patient is then still thyrotoxic the dose can be repeated. If she is myxoedematous, she will need thyroid treatment for life.

There is a theoretical risk of carcinoma developing in the irradiated gland. Although this has never yet been reported after radio-iodine, this treatment is not usually given to young patients.

At present, only a few hospitals are equipped to handle radio-active substances.

Surgical treatment is indicated when the patient is under 50 and has a large, unsightly goitre, or when medical treatment has failed or has caused toxic symptoms. It is often the best treatment in other cases, avoiding the prolonged supervision necessary in those treated medically. For about ten days before the operation the patient should be at rest and mainly in bed. A calm, reassuring atmosphere is most important for these nervous patients. Lugol's iodine is given by mouth for ten days before the operation; this helps to restore the thyroid activity to normal. Some surgeons prefer all symptoms of thyrotoxicosis to be controlled by anti-thyroid drugs before operation, Lugol's iodine being given as well for the last ten days. If auricular fibrillation is present, digitalis is given.

In expert hands the mortality of the operation is below 1 per cent., the scar quite inconspicuous and the results excellent. All symptoms except exophthalmos are relieved in successful cases. Relapses, however, may occur. If too much of the gland is removed myxoedema results, and will require thyroid treatment for the rest of the patient's life.

Treatment of Thyrotoxic Crisis.—This serious complication requires

intensive treatment and first class nursing. Large doses of a *sedative* are necessary (e.g. chlorpromazine). For high fever, *tepid sponging* may be required. *Iodine* is given by mouth or intravenously, and an *anti-thyroid drug* such as methyl thiouracil. *Copious fluids* are necessary, by mouth or intravenously or both. *Steroids* are given in some cases, as adrenal failure (p. 235) may occur.

Case Histories

(1) Mrs. B., *aged 22*, a housewife. She became nervous and irritable, breathless and lost a lot of weight following the birth of her second baby. Refused to see her doctor or attend post-natal clinic. She had her photograph taken to send to her mother in Australia and herself noticed a slight goitre in the picture. She went to her doctor who sent her to hospital. She was admitted immediately. Emotional and agitated; slightly enlarged soft thyroid gland; pulse rate 130; B.M.R. +40. Thyroidectomy after three weeks pre-operative treatment with methyl thiouracil. She then went to a convalescent home. Improved rapidly and gained 2 stone in weight.

(2) Mrs. J., widow, *aged 60*. She had had auricular fibrillation for several years, thought to be due to mild hypertension. One day she walked into the surgery to ask for a further supply of digitalis tablets; to her doctor's astonishment her pulse rate was 180. She was not very breathless. The dose of digitalis folia was increased from 2 to 3 grains a day, but the rate was not controlled and coupled beats appeared. She was sent up to hospital where investigations showed that she had thyrotoxicosis, though she had no other symptoms of thyroid trouble. After a single dose of radio-iodine she improved markedly, and six months later said she had not realised how tired and nervy she had been before treatment.

(b) Simple Goitre

Here the large gland makes the neck lumpy but the patient is otherwise well. In many districts—for instance, parts of Switzerland, India and Derbyshire—goitre is *endemic*, i.e. widely prevalent among the local population. This is because the water contains *too little iodine*; as thyroxine contains iodine the gland works desperately to produce bricks without straw and becomes enlarged in so doing. Goitres may arise from other causes than lack of iodine. As in other thyroid disorders, nearly all those affected are women. The enlargement of the gland becomes noticeable during adolescence.

Women with simple goitre occasionally develop thyrotoxicosis, myxœdema or cancer of the thyroid.

Treatment.—If the lump is very ugly, or if it is pressing on the windpipe, or if signs of thyrotoxicosis or cancer develop the gland should be removed. Thyroid treatment may be required after operation.

In some non-endemic cases small doses of thyroid are given to adolescents.

Prevention.—In goitrous districts the addition of iodine to cooking and table salt (one part in 100,000) prevents the development of goitre.



Fig 56 A woman with a large Nodular Goitre

(c) Thyroiditis

Acute thyroiditis is rare. It may occur as a complication of mumps, or other infection, or with no obvious cause. The gland suddenly becomes enlarged and tender, and the enlargement lasts for days or weeks. No effective treatment is known.

Hashimoto's thyroiditis is more common. The thyroid gland is enlarged and there are symptoms of myxœdema. The *cause* of the disease is a strange one—the patient develops antibodies to his own thyroglobulin (a protein present in the normal thyroid) just as if it were a foreign invader like an organism. The result is that he develops symptoms of thyroid lack (myxœdema). These respond to

thyroid treatment. Operation is only necessary if the thyroid gland causes pressure symptoms.

(d) Myxœdema

In myxœdema the thyroid gland no longer functions properly, instead of being overactive, and many of the symptoms are the exact opposite of those of thyrotoxicosis. Myxœdema may occur in

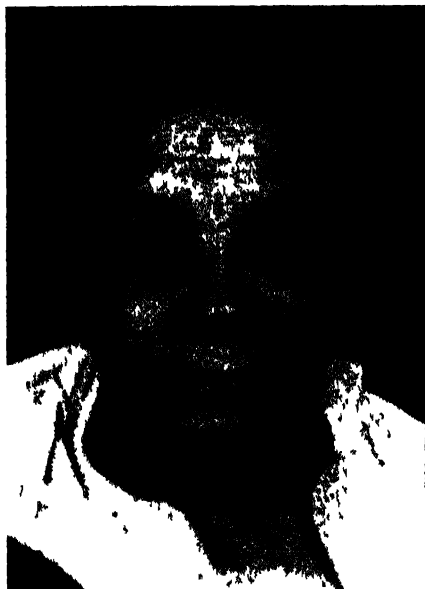


Fig. 57. Advanced Myxœdema. Patient died a few days after admission. (Dr. Richard Asher's case)

patients with simple goitre or with Hashimoto's thyroiditis; at other times it is the result of the over-enthusiastic treatment of thyrotoxicosis; often there is no apparent cause for its occurrence.

Myxœdema is most common among middle-aged women. The symptoms creep on by such slow degrees that nothing abnormal may be noticed for several years until the thought suddenly strikes the patient or her relatives: "This woman has changed." She has become slow, torpid and inert in thought and movement; she feels the cold and sits huddled over the fire. Her heavy, dull face, which expresses little emotion, is pale, with baggy eyelids and thickened features; the skin is faintly yellow and is dry and coarse in texture. The hair and eyebrows tend to fall out. Some myxœdematous patients suffer from heavy periods; joint pains are also common.

There are characteristic swellings of the hands and feet, and over the clavicles. The patient is often deaf, which adds to the apparent stupidity; her voice is harsh and expressionless. She eats little, yet puts on weight; her pulse is slow, her temperature subnormal and her bowels constipated. Advanced myxœdema is unmistakable, but nothing is easier to miss than an early case. Mental symptoms may be so prominent that the patient may be certified and admitted to a mental hospital.

Investigation shows that the B.M.R. is lowered to -15 or less, and the patient is usually anæmic. The *serum cholesterol* is raised. The *E.C.G.* shows characteristic changes.



Fig 58. Myxœdema developing after thyroidectomy—before and after treatment (Dr Richard Asher's case)

Treatment.—There are few more gratifying experiences than to witness the successful treatment of myxœdema. We see a heavy, unresponsive sluggard, mentally retarded to the point of insanity, transformed into a human being. This transformation is wrought by thyroid extract. Very small doses are given at first (e.g., $\frac{1}{4}$ gr. twice a day), as larger doses may precipitate heart failure. The dose is gradually increased to $\frac{1}{2}$ –1 gr., two or three times a day. The patient must take thyroid for the rest of her life or she will inevitably relapse into her former state. The dosage is regulated by the patient's condition and by her pulse rate, which should not rise above 75 at rest.

Although the results of thyroid treatment in myxœdema are so striking, the patient's relatives should be warned that complete

restoration of her original personality is not always possible, and that she may still be rather more slow-witted than she was before her illness.

Myxoedema Coma

This complication carries a high mortality; it is a serious medical emergency. The patient sinks from apathy into stupor and from stupor into coma. Her skin feels cold to the touch, like that of a fish. The *temperature* is low—sometimes as low as 80° F. (A special low-reading thermometer is necessary—always use one if the temperature of a cold unconscious patient does not register above 95° on the ordinary thermometer.) The *pulse rate* is also low—perhaps as low as 30. The *blood sugar* is often low too.

Treatment.—The patient must be nursed in a warm ward, well supplied with warm bed clothes and hot water bottles. The most rapidly acting thyroid-like substance available—*triiodothyronine*—is given. *Glucose* is given intravenously. *Steroids* are necessary too. *Antibiotics* are given because these patients are very susceptible to infection.



Fig. 59. Cretin, before and after treatment. (A cretin left untreated as long as this one would be unlikely to develop normally.)

If the patient recovers, thyroid extract is gradually substituted for triiodothyronine.

Case History.—Miss B., aged 70, an old rheumatic cripple, had been visited by her doctor intermittently for several years, until one day, seeing her after a gap of some months, he suddenly said to himself “Myxoedema.”

She had the characteristic coarse features, falling hair, slow pulse and corn-crake voice; she was almost stone deaf, but her mental powers were adequate. After a month on thyroid gr. $\frac{1}{4}$ thrice daily she was unrecognisable. Her features had fined down, her colour and complexion had improved and she was not so deaf. She herself was not in the least impressed and asked not to be visited again to save expense (this was in the days before the National Health Service). She was urged, however, to continue to take thyroid, and this she promised to do.

(e) Cretinism

A cretin is a baby born without a thyroid gland, or with one which does not function. A mother with a simple goitre due to iodine deficiency may give birth to a cretin. Cretinism is equally common among males and females, unlike other thyroid diseases.

An observant mother or nurse will notice that there is something wrong with a cretinous baby about the age of three months, or even earlier. The baby does not smile or try to lift his head, he feeds reluctantly, his tongue seems too big for his mouth, his eyes are too far apart, his cry is hoarse and he is constipated. The skin is wrinkled and looks as if it were too big for his body; often there is an umbilical hernia. If he is not treated his backwardness and cretinous appearance will soon become more obvious.

Untreated cretins grow up to be imbecile dwarfs. Every stage of the child's development is delayed. A cretin is stunted, thick-set, with pendulous abdomen and a crooked spine. The face is pale and pasty, with a broad, flattened nose, thick lips and a lolling, cracked tongue. The child is often deaf and can seldom talk much owing to the mental defect. Sexual maturity is seldom attained. Thus we see that the thyroid secretion is essential to normal physical, mental and sexual development.

Treatment.—Thyroid extract must be given as soon as the cretinism is diagnosed and continued throughout life. If the diagnosis is made and treatment begun in the first few months of life there is a good hope that physical and mental development will be normal. All too often the diagnosis is delayed until the child is a year or two old; thyroid treatment begun at this age will improve physical growth but the child will be mentally defective. The vital importance of early diagnosis and treatment must never be forgotten. A nurse or health visitor might be the first person to whom the mother confided her fears. Although very few backward babies are cretins, the possibility must be borne in mind. The tragedy of the missed case, untreated until it is too late, will then be avoided.

(2) Pituitary Disorders

The pituitary gland is placed below the brain, about two inches behind the eyes. This remarkable structure, scarcely bigger than a



Fig 60 Cretin aged 3½ months.
Note characteristic face, umbilical hernia and wrinkled skin
(From Fanconi and Wallgren's *Textbook of Paediatrics*, Heinemann)

pea, produces about a dozen hormones and controls the thyroid, the sex glands and the adrenal cortex.

The pituitary gland, though so tiny, is really made up of two quite separate glands. The *posterior pituitary* produces two hormones. One of these causes *contraction of the pregnant uterus*. An extract of posterior pituitary (for instance, pitocin) is to be found in every midwife's bag. The other hormone (anti-diuretic hormone) *raises the blood pressure* and prevents *too much urine* being formed.

The *anterior pituitary* produces at least six hormones. Some of these hormones control the *thyroid gland*, and the *adrenal cortex*, and the *sex glands*. (A.C.T.H.—adrenal corticotrophic hormone—is an extract of the hormone controlling the adrenal cortex.) Through the hormones which control ovarian secretions, the anterior pituitary regulates the menstrual cycle and helps to control pregnancy. Other pituitary hormones *induce lactation*, *stimulate growth*, and help to regulate *the body's sugar supplies*; diabetes mellitus may well be first and foremost a pituitary disorder. Excess or lack of some of these hormones produces the disorders described below.

(a) Diabetes Insipidus

Here there is a shortage of the *anti-diuretic hormone* of the posterior pituitary. The disease may follow meningitis, injury or tumour of the pituitary; sometimes no cause can be found. The patient passes several gallons of urine daily and is always thirsty. Symptoms can be controlled by giving posterior pituitary extracts, either by injections or in the form of snuff. Treatment usually has to be continued indefinitely.

(b) Gigantism

A circus giant may have suffered from *too much growth hormone* while he was growing. An overgrowth or tumour of the anterior pituitary may be present which may require surgery or X-ray treatment. Giants may develop acromegaly in later life.

(c) Acromegaly

Here the over-production of growth hormone occurs in an adult who has stopped growing. The acromegalic has large hands and feet, and the features are distorted by enlargement of bones of the face and jaw. The soft tissues are over-developed too; thickening of the tongue makes speech sluggish and the enlarged larynx results in a deep voice.

Acromegaly is often the result of a tumour of the anterior pituitary, and the tumour itself may cause symptoms such as headaches, or visual symptoms due to pressure on the optic nerves, which lie just

above the pituitary. Other pituitary functions are sometimes disturbed, leading to diabetes mellitus, sexual changes or mental deterioration. If the tumour involves the posterior pituitary, diabetes insipidus may occur.

X-ray or surgical treatment is sometimes successful.

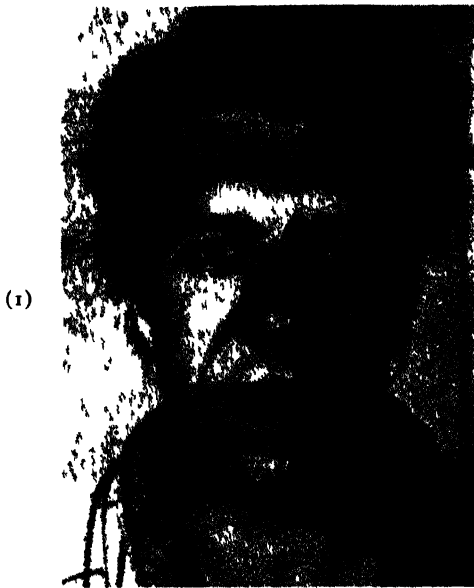


Fig. 61 Acromegaly

1 Showing enlargement of features 2. Same patient's hand, photographed beside a normal woman's hand. (Dr Richard Asher's case)

(d) Infantilism (Loraine Syndrome)

This causes the *Peter Pan* type—small, dainty, and emotionally and sexually immature. Probably growth- and sex-stimulating hormones are both defective. Treatment with anterior pituitary extracts may be successful.

(e) Simmonds' Disease (Panhypopituitarism)

In this condition, disease or destruction of the anterior pituitary affects the production of *all* the pituitary hormones. Thrombosis or embolism of the artery supplying the gland is probably the commonest cause; many cases follow severe post-partum hæmorrhage. Tumours, fractures of the base of the skull, tuberculosis and syphilis account for a small proportion of cases; sometimes no cause can be found.

Symptoms come on gradually. Following a post-partum hæmorrhage, for instance, the patient's menstrual periods do not return. She ages rapidly, her skin becomes wrinkled, and pubic and axillary hair become scanty. She feels cold and her temperature is probably subnormal. She eats little, feels weak and is mentally slow. Her blood sugar is often low. In severe cases the patient goes into coma; coma may be precipitated by an acute infection, or by sedatives, or by exposure to cold, or by water logging. The patient is pale, cold, and her temperature is low, as in myxædema coma (p. 225). The blood sugar is also low—and this can cause coma (p. 245). The mortality in such cases of coma is high.

It will be seen that the patient's symptoms can be accounted for by inactivity of the sex, thyroid and adrenal glands, for these glands cannot function normally without the stimulus of the anterior pituitary hormones.

Investigations.—An *X-ray of skull* may show changes characteristic of a tumour in the neighbourhood of the pituitary.

Ketosteroid Output.—The ketosteroids are substances derived from the adrenal cortex which are normally excreted in the urine. In Simmonds' disease ketosteroid output is abnormally low, because adrenal cortical activity is low.

Treatment.—Steroids in small doses, or A.C.T.H., are given. Small doses of thyroid and testosterone (male sex hormone) are often given as well. If a tumour is present, surgery or X-ray treatment will probably be needed.

Treatment of Coma.—The patient is allowed to warm up slowly. Intravenous glucose is given to correct the low blood sugar, and

intravenous hydrocortisone hemisuccinate to supply the missing adrenal cortical hormone.

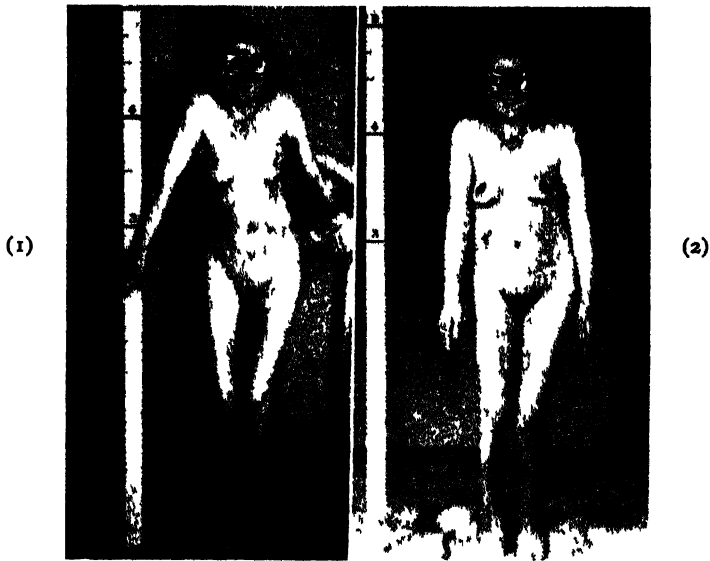


Fig 62. Simmonds' Disease.

The illness was caused by thrombosis of the pituitary, following birth of baby twenty years previously. 1. Showing wasting, weakness, senile changes, absence of pubic hair, etc. 2. After treatment with testosterone and small doses of stilboestrol and thyroid. (Dr Richard Asher's case)

(3) Diseases of the Adrenal Gland

Like the pituitary, the adrenal has two parts—in this case *cortex* and *medulla*. The adrenal cortex (the outer part of the gland) produces hormones which are necessary to life. They regulate the water and salt content of the body, and are concerned with carbohydrate and protein metabolism, and with sexual function. Some of these hormones (e.g. cortisone and hydrocortisone) are used clinically.

The adrenal medulla produces *adrenaline* and related substances. Adrenaline raises the blood pressure, quickens the pulse, constricts small blood vessels (p. 269), relaxes bronchial spasm (p. 136), raises the blood sugar (p. 240) and stimulates the sweat glands.

Diseases of the Adrenal Cortex

These may result either from an *excess* or *lack* of adrenal cortical hormones; they are all rather uncommon. There may be an excess of hormones because there is an adrenal cortical *tumour*, or because the glands themselves are *over-active*. Some tumours of the anterior

pituitary cause symptoms of cortical over-activity because they produce an excess of A.C.T.H. which overstimulates the adrenal cortex. Symptoms of adrenal cortical over-activity depend on which set of hormones is mainly concerned—the group regulating metabolism, or the group affecting sexual function. However, the distinction is not clear cut; a patient may have symptoms of both types of disturbance.

Cushing's Syndrome

Here the hormones controlling water, salt, sugar and protein metabolism are mainly involved; many of the symptoms are similar to the side-effects of cortisone treatment (p. 60). The patient becomes fat, round shouldered and moonfaced. Purplish streaks are to be seen on the abdomen and thighs (similar to the *striae gravidarum* which develop in pregnancy). Backache is common, and X-rays show that there is osteoporosis, sometimes leading on to compression fractures of the vertebræ. This is due to *loss of protein* from the bones, and loss of protein from muscle makes the patient *weak*. Sugar is often found in the urine, and diabetes mellitus may develop. Oedema and hypertension are common. The patient may die of some infection to which he is susceptible.

Case Histories

1. Mrs. X aged 45. Depression and paranoia (see p. 397) for two years. She was admitted to a mental hospital; mute; refused food; had to be forcibly fed. She had amenorrhoea, growth of facial hair and spontaneous bruising. (No obesity because of lack of appetite). While in the mental hospital she developed foot drop as a result of an injection of paraldehyde into the sciatic nerve. Transferred to a general hospital for treatment of the foot drop. Cushing's syndrome diagnosed; adrenalectomy performed. Complete recovery, both physical and mental (Fig. 63). Remains well and happy.

2. Mrs. Y., aged 39. For five years she had a disabling, obsessional neurosis (see p. 405) which obliged her to spend most of the day washing and cleaning. She realised her behaviour was abnormal and sent her 5 year old daughter to be brought up elsewhere to keep her away from her "silly ideas". A leucotomy did not help. Later she was referred to a general hospital; Cushing's syndrome was diagnosed and adrenalectomy performed. All mental symptoms cleared up and the patient's appearance was transformed (Fig. 64). She took her daughter back and soon after had another baby. Remains well and happy.

Adrenogenital Syndrome

Certain adrenal cortical hormones are responsible for masculine characteristics such as low voice, beard growth and pubic hair of



Fig 63 Cushing's Syndrome (1) Before and after adrenalectomy. Note facial hair and dilated blood vessels in pre-operative photograph (See p 232 for case history) (Dr Richard Asher's case)



Fig 64 Cushing's Syndrome (2) Before and after adrenalectomy. Pre-operative photograph shows facial hair, dilated blood vessels and obesity (See p 232 for case history) (Dr Richard Asher's case)

male distribution. An excess of these hormones results in the adreno-genital syndrome. Female patients become masculine, and young boys reach puberty early.

Adrenogenital syndrome may be due to tumours, which are sometimes highly malignant.

Investigations.—The urinary ketosteroid output is always raised.

Treatment.—If a *tumour* is present it should be removed. If there is no tumour, but the glands are over-active, both adrenals may have to be excised. The patient will then need cortisone treatment for life.

Addison's Disease

In this rare disease there is a *lack* of adrenal cortical hormones, due to disease of the adrenal glands—often tuberculosis.

Symptoms.—The patient with Addison's disease is usually *thin, weak and brown*. The skin pigmentation is very characteristic; there is generalized tanning with darker patches over pressure areas such as knees and knuckles. The patient commonly suffers from symptoms such as nausea, vomiting, abdominal pain and diarrhoea. He feels cold and his temperature is low; his pulse is feeble and his blood pressure low.

Addisonian crises are acute exacerbations during which the patient is prostrated by severe vomiting and diarrhoea, and is dehydrated and collapsed.

Investigations

(1) The blood sodium and chloride are low, whereas there is an excess of salt (sodium chloride) in the urine.

(2) The blood sugar is often low.

(3) The urinary ketosteroids are low, even after an injection of A.C.T.H.

(4) If the patient drinks a large amount of water, diuresis does not occur promptly (Kepler test).

All these tests show that the adrenal cortex is functioning poorly, for the various adrenal cortical hormones help the body to retain salt, raise the blood sugar, or are excreted as ketosteroids in the urine.

(5) X-ray of abdomen sometimes shows up calcified adrenals in tuberculous cases.

(6) An X-ray of chest is necessary if tuberculosis is suspected.

Treatment.—Steroid treatment replaces the missing hormone and maintains life and (often) health. Cortisone (25–40 mgm. daily in divided doses) is usually given; during an infection or operation the dose must be increased. Sometimes extra *salt* is needed. In addition to cortisone D.O.C.A. (dexoxycorticosterone acetate), or fluoro-hydrocortisone may be given; they are specially effective in enabling the body to retain salt.

Acute Adrenal Insufficiency.—The symptoms are those of the Addisonian crises described above—vomiting, diarrhoea, weakness, dehydration, and collapse, with pallor, thready pulse and low blood pressure. Acute adrenal insufficiency occurs not only in Addison's disease. It may occur in any severe infection or septicæmia (e.g. meningococcal septicæmia, p. 324) due to hæmorrhage into the adrenals. An increasing number of cases occur as a complication of steroid therapy. The mechanism is as follows: when large doses of steroids are given over a long period, the patient's adrenal cortex stops functioning, and ceases to produce natural cortical hormones. This means that the gland cannot supply the increased amounts of cortical hormones which the body needs during acute illnesses or operations. Unless increased doses of steroids are prescribed at such times for patients on steroid treatment—or *who have recently received such treatment*—there is a grave risk of acute adrenal insufficiency. Hence it is vital to know whether patients have had steroid treatment before admission to hospital for an illness or operation.

Treatment.—Intravenous hydrocortisone hemisuccinate and intravenous glucose are given.

Diseases of the Adrenal Medulla

These are all rare. The only one which need be mentioned is *phæochromocytoma*. Although this tumour of the adrenal medulla is rare, it is important because it causes *hypertension*, and in cases of severe hypertension—especially in young people—phæochromocytoma may be suspected, and nurses will often see investigations carried out to exclude this disease.

Symptoms.—The patient suffers from hypertension, either continuous or occurring in paroxysms. During these paroxysms severe headache, palpitation, anxiety, blurred vision, and other symptoms occur; the patient is seen to sweat profusely, his skin is cold and pale and the blood pressure is very high (300 mgm. systolic or more).

These symptoms are caused by the excess of adrenaline and allied substances poured out by the tumour.

Investigations.—(1) There is an excess of catecholamines in the urine.

(2) An injection of phentolamine ("rogitine")—a substance which antagonises adrenaline—produces a fall in blood pressure.

If this test is being carried out the patient must be resting quietly but *must not be given a sedative*, as this makes the results unreliable.

Treatment.—It is often possible to remove a phæochromocytoma

and so cure the disease. Hence the importance of this rare disease—it is one of the few curable causes of hypertension.

(4) Diseases of the Parathyroid Glands

There are four parathyroid glands; they are about the size of a pea and are embedded in the back of the thyroid gland. They produce a hormone called *parathormone*. This hormone regulates the amount of *calcium* and *phosphorus* in the blood. If there is *too little parathormone* the blood calcium falls; this makes the nerves over-excitabile and *tetany* results. *Too much parathormone* has the opposite effect; the extra calcium comes from the skeleton, where calcium salts give the bones their normal hardness. The bones become soft and deformed as their calcium salts drain away. (*Osteitis fibrosa cystica*.)

Tetany may follow thyroidectomy, the parathyroids having been inadvertently removed along with the thyroid. Tetany occurs in any condition in which the blood contains too little calcium. Thus, it may occur in *steatorrhœa* (p. 148) because calcium is poorly absorbed from the bowel. Calcium absorption is also defective in *rickets*, and here, too, tetany may be seen. Tetany can also be caused by *alkalosis*, after an excess of alkaline drugs. Repeated vomiting may cause alkalosis and tetany, because so much acid gastric juice is lost in the vomit. *Hysterical overbreathing* may have the same effect. This is because overbreathing washes too much carbon dioxide (CO_2) out of the blood. CO_2 in solution is slightly *acid*, so that its removal makes the blood more alkaline—hence the tetany.

The patient with tetany suffers from repeated painful cramps and spasms of the muscles of hands and feet. *Spasm of the larynx* causes crowing respiration. Tetany usually responds quickly to injections of calcium salts; parathormone injections are sometimes given too. The acute stage once passed large doses of calcium and vitamin D are given, and the cause of the tetany, where this is known, should be treated.

DIABETES MELLITUS

THIS remarkable disease is of special importance to doctors and nurses. There are about 200,000 diabetics in this country, and all these men, women and children will need treatment throughout life. Success in treatment depends on a thorough understanding both of the disease and of the patient, for no two diabetics are alike, and treatment must be made to measure in each case.

Nurses play a most important part both in the diagnosis and treatment of diabetes. As already mentioned (p. 29) the nurse may make the diagnosis by finding sugar in the urine when diabetes is unsuspected. While the patient is in hospital she carries out the crucial urine tests, on the results of which treatment depends. She often helps to prepare the carefully weighed meals. Finally she helps to teach the patient about his disease and its management, so that he will be able to take control when he leaves hospital. To do all this an understanding of the nature of diabetes is necessary.

In this chapter the symptoms and signs of diabetes are first described; then we shall want to know what causes these symptoms and signs, after which it will be easy to understand the treatment of diabetes. The chapter ends with an account of diabetic complications.

Symptoms of Diabetes (Mild and Severe)

There are two types of diabetes. The commoner variety is a rather mild disease of insidious onset, affecting chiefly stout middle-aged and elderly people. The patient may complain of general ill health, gain in weight or irritation (pruritus) of the genitals. Often the first symptom is that of one of the complications of diabetes—carbuncle, cataract, neuritis or gangrene of the toe. Or there may be no symptoms at all, the diagnosis being made on finding sugar in a routine specimen of urine. *Coma* is rare in this type of diabetes.

The less common variety of diabetes is a much more severe disease, usually affecting young people. Symptoms may start quite suddenly. The patient is tormented by thirst, he drinks avidly and passes quantities of urine daily—often two gallons or more. The urine causes pruritus. The patient loses weight—sometimes several stone—and he feels tired and weak. *Coma* is a common complication in this type of diabetes—indeed the patient may be in coma when the diagnosis

is first made. (Infection often precipitates the coma, both in the treated and the untreated case.)

Diagnosis

In only a small proportion of cases can the diagnosis be made with any confidence on clinical examination alone—namely, in cases of acute diabetes in young people. In all other cases urine tests will be necessary, and often blood tests as well.

The Urine in Diabetes—Urine testing is absolutely necessary not only for the diagnosis of diabetes but also for the control of the disease throughout the patient's life. So it is most important to understand the meaning of the tests one has to do so often.

Everyone knows that diabetic urine contains sugar (glucose). Benedict's test (less often, Fehling's) is used as a test for this sugar in the urine. So the first question is: Does a positive Benedict's test indicate diabetes? The answer is No, but a *red precipitate* in this test is so rare in any other condition that such a result makes the diagnosis of diabetes very likely; if the tests for *acetone* are also positive we can be practically certain the case is one of diabetes.

What is the diagnosis if Benedict's test results in a green solution with a slight yellow or orange precipitate, and acetone tests are negative? In a young patient such a result is unlikely to indicate diabetes if there are no symptoms of this disease, but in elderly people, in whom diabetes comes on so gradually, a moderate amount of glucose in the urine is more suspicious.

Glycosuria also occurs in meningitis, head injury, cerebral and sub-arachnoid hæmorrhage, pituitary and thyroid disease, emotional excitement, and in people who eat too much starchy food. In all these conditions there may be so much sugar in the blood that some of it leaks out into the urine.

Then there are some people who leak sugar into the urine even when there is no excess of sugar in the blood (i.e. blood sugar below 180 m.gm. per 100 ml. blood). These people are said to have a *low renal threshold*, because the kidney lets sugar pass the "threshold" even when the blood sugar is quite low. This condition is quite harmless; blood tests are often necessary before we can be sure the case is one of a low renal threshold and not diabetes.

The urine also gives a positive reaction with Benedict's during the later months of pregnancy; in this case the sugar is not glucose but *lactose* (milk sugar) which reacts in the same way with Benedict's solution.

To sum up: whenever a small amount of sugar is found in the urine it should be reported and further investigations may be necessary; diabetes may or may not be the cause. But a brick-red Benedict's test *practically always means diabetes*.

Tests for Acetone.—Acetone and its relatives are collectively known as *ketones*. When the blood contains too much ketone (*ketosis*) these substances appear in breath and urine. Acetone in the breath smells like nail-varnish remover, and is unmistakable when once smelt. (It is a curious—and unfortunate—fact that some people are quite unable to detect the smell of acetone.) Acetone and other ketones in the urine can be detected by means of two tests—Rothera's nitro-prusside test and the ferric chloride test. In Rothera's test a magenta colour appears even if the merest trace of ketone is present; ferric chloride only produces a violet colour in severer degrees of ketosis.

Ketosis occurs not only in diabetes but in many childish fevers and digestive upsets, and after prolonged starvation or vomiting. In a suspected case of diabetes the diagnosis is confirmed if acetone is found in the urine; in a known diabetic such a finding is a sign of threatened coma.

Specific Gravity Tests.—Finally, sugar in the urine makes the urine heavier, so that the specific gravity is often 1030 or more, instead of the normal 1010–1020. But this test is quite unimportant compared with the chemical tests for sugar.

Blood Sugar Tests.—The blood sugar is always raised at some time of day. Normally the blood sugar varies between 80 and 180 m.gm. per 100 ml., being higher after a meal. In severe diabetes the fasting blood sugar may be several hundred m.gm. per 100 ml., rising still higher after food.

In doubtful cases a *glucose tolerance test* is sometimes necessary. This test shows how the body deals with its sugar supplies. The patient's blood sugar and urine are tested before breakfast, and he then drinks a solution of 50 gm. of glucose; further blood and urine tests are then done at half-hour intervals. The normal person's blood sugar rises after a large dose of glucose, but that of a diabetic rises much higher and takes longer to come down. A glucose tolerance test is unnecessary and may be dangerous in an obvious case of diabetes.

Cause of the Diabetic State

The diabetic's symptoms and the changes in the urine are all due to the failure of the body to deal normally with its *carbohydrates*.

Carbohydrates make up the body's chief supply of fuel, and their combustion provides it with energy, just as petrol drives a motor engine. In diabetes both *fuel storage* and *fuel consumption* are upset. Carbohydrate supplies are taken in from the food as in the healthy person; starch and sugar are digested in the intestine and the glucose so produced is absorbed into the blood stream. But in the diabetic the *fate* of this glucose is abnormal. In a normal person some of the absorbed glucose is stored in the liver as a starch-like substance called *glycogen*, some is stored in the muscles, some is burnt and a little overflows into the blood stream. Hence, the blood sugar normally rises after a meal (from about 80 to 180 m.gm. per 100 ml.). In between meals the liver keeps the blood sugar from falling too low by releasing glucose from its store of glycogen. The blood supplies the tissues with the sugar they need to supply them with energy. The muscles use up a great deal of sugar, especially during exercise.

The blood sugar is regulated by six hormones, which are so exquisitely and harmoniously adjusted that the most violent exercise, prolonged fasting or the largest banquet can only alter the total amount of sugar in the whole blood stream by about a teaspoonful. The most important of these hormones is, of course, *insulin*, manufactured by some special cells in the pancreas. This insulin affects both the *storage* and *consumption* of glucose. Insulin enables the liver and the muscles to store glycogen, and it enables all tissues to burn glucose; it also stops the liver from breaking down protein - another process which liberates glucose. Thus all the actions of insulin tend to *reduce* the amount of sugar in the blood (in other words, insulin *lowers* the blood sugar). Insulin is antagonised by five other hormones, produced by the pituitary, thyroid and adrenal glands, all of which tend to *raise* the blood sugar. After a large meal the pancreas produces more insulin to deal with the extra glucose; insulin production falls during a fast. If muscular exercise calls for more glucose the adrenal glands produce more *adrenaline* which speeds up the release of glucose from liver glycogen.

✓ In the diabetic this beautifully balanced arrangement is entirely disorganised by the failure of the body's insulin supplies. The glucose absorbed during a meal, instead of being quickly stored or burnt, circulates unchanged in the blood. The tissues, though bathed in sugar, cannot use it, and the patient starves in the midst of plenty, like the Ancient Mariner who described "Water, water, everywhere, nor any drop to drink." The liver recklessly adds to the confusion by breaking down protein as well as glycogen, liberating still more glucose. Thus there is so much glucose in the blood that the

kidneys cannot dam it back and some appears in the urine. The sugar passed by the kidney draws water after it so that large volumes of urine are passed. The patient drinks avidly to make up for this loss of water in the urine, and so we come back to the typical symptoms of diabetes—thirst, polyuria, loss of weight and sugar in the urine. ✓

Even this is not the whole story. The diabetic, unable to burn sugar, falls back on the body's emergency fuel supply and burns *fat*. But the body is unused to burning so much fat, and so this emergency fuel is only partially consumed. The products of this incomplete fat combustion are the *ketones* which thus accumulate in the blood and appear in breath and urine. And ketones, rather than excess sugar, cause diabetic coma.

The *fundamental causes* of diabetes are mostly unknown. The disease is sometimes hereditary; about one case in four has a family history of diabetes.

The Treatment of Diabetes

In the old days, before Banting and Best discovered insulin, all that could be done for the diabetic was to put him on a starvation diet, giving him as little as possible of the carbohydrate that his body cannot use. This often prolonged life, and fat, elderly diabetics with mild diabetes can still be kept in health on a suitable diet without insulin. In such cases we must assume that the patient can produce enough insulin to cope with a reasonable amount of sugar but not with a glutton's diet. But severe diabetics produce so little insulin of their own that they cannot deal with the most restricted diet unaided. These people were doomed to death before 1922; they are the sort who need insulin to-day.

(a) The Treatment of Mild Diabetes

Fat, elderly diabetics with no sign of ketosis can generally be treated simply by *regulating the diet*. A loss of weight and a marked improvement in general health often follows. *Insulin is seldom necessary in such cases.*

A *low carbohydrate diet* is necessary. Sugar and all sweet foods are forbidden or drastically reduced, and starchy foods such as bread, puddings, rice, pastry, and potatoes are also restricted. *Fats* are allowed in moderation; *protein foods* are unrestricted (p. 253).

If the patient keeps strictly to such a diet she will lose a considerable amount of weight. Usually she feels much better, and tests

show that the blood sugar is lower and glycosuria has been reduced or abolished. Once her weight has returned to normal the diet may be increased, but sugars and starches should still be restricted.

(b) Treatment of Moderately Severe Diabetes in Elderly Patients

If the patient still has heavy glycosuria after her weight has returned to normal, or if she is of normal weight at the start of treatment, antidiabetic drugs will probably be necessary. Either *insulin* or *oral drugs* may be given; as a rule oral drugs are tried first.

Treatment of Diabetics with Oral Drugs.—These drugs are usually effective only in the middle aged and elderly diabetic; they are never given to young patients or those with ketosis. Their mode of action is not fully understood. *Tolbutamide* is the most commonly used; the usual dose is 0.5 to 1 gm. twice daily, but larger doses may be given. *Dibetin* is another compound which may be useful.

(c) Treatment of Severe Diabetes

Young patients and those with *ketosis* always require insulin.

Diet.—These patients are always thin and need a generous diet. Proteins and fats are unrestricted, and a fairly generous ration of carbohydrate is allowed (e.g. 150–250 g. a day). *Unlimited* sugar and starch is not advisable, because it would then be impossible to control the blood sugar with one or two doses of insulin a day. (It has been found that complications of diabetes are more common when the blood sugar is inadequately controlled.)

As a rule the patient is given a diet sheet which tells him how much of each food contains 10 gm. of carbohydrate. For instance, a piece of bread $4 \times 3 \times \frac{1}{2}$ inch, a potato the size of an egg, a dessertspoonful of jam or sugar, or three tablespoonfuls of unsweetened stewed apple each contain 10 gm. carbohydrate. Suppose the diet is to contain 250 gm. carbohydrate a day, the patient selects 25 of these portions, dividing them up between the three main meals.

Insulin Treatment.—Our aim is to replace the patient's deficient insulin supplies with injected insulin, to keep the blood sugar as far as possible within normal limits, and to do this, if possible, with one daily injection. There are several types of insulin available. *Soluble insulin* acts quickly and for a short time and is essential for the treatment of ketosis and coma. By itself it is unsuitable for the daily treatment of a stabilised diabetic, for its effects last less than 12 hours, so that two or three daily injections would be necessary. All the other insulins come into action more slowly and act for longer periods (up

to 30 hours). This delayed action enables most diabetics to be stabilised with one injection a day. Delayed action insulins include zinc protamine insulin (Z.P.I.), globin insulin and the insulin zinc suspension mixtures (such as insulin lente).

When insulin treatment is first started the right dose has to be found by trial and error. A moderate dose of one of the delayed action insulins is given half an hour before breakfast—e.g. 20–30 units Z.P.I. or insulin zinc suspension. If the case is a severe one larger doses will be necessary, often combined with soluble insulin, which takes care of the breakfast carbohydrate before the other has come into action. In very severe diabetes a second injection (of soluble insulin) is necessary in the evening, before supper. Fortunately, few diabetics need two daily injections. Diet and insulin must both be adjusted so that the patient is kept in good health, at a proper weight, on a diet which satisfies him and which will fit in with his ordinary everyday life; finally, urine and blood tests must show that his diabetes is under control.

When first treating a diabetic we may have to test the urine every few hours—one of the heaviest tasks in a diabetic ward. These tests are the essential guides to treatment, and they enable us to follow with great accuracy the battle between disease and treatment, and so are of unusual interest. In severe diabetes we do not aim at sugar-free specimens throughout the 24 hours, or the patient will be in danger of insulin overdosage at certain times of day. All specimens should ALWAYS be acetone-free.

Urine Charts.—When filling in the patient's urine chart it is best to enter the results of the Benedict's test according to the colour obtained—red, yellow, green or blue—rather than to write up "Sugar ++" etc., which may mean one thing to one person and something else to another. Diabetic charts often have the diet and insulin entered as well as the urine tests, which is a very good plan. And, finally, let it be repeated, *DON'T* record the volume of urine in Roman figures, for your own sake and that of the readers of your records.

Training the Diabetic

Suppose the diabetic has been stabilised on a satisfactory regime of diet and insulin and is now ready for discharge from hospital. (The milder cases can be treated as out-patients throughout.) Before he goes it is usually the nurse's job to explain the diet chart and to teach him how to give himself injections and to test his urine. ("Clinitest" is generally used, and the urine tested three times a day.) This may be a difficult task, but it is a crime to discharge a patient

from the sheltered backwater of a hospital ward into the hurly-burly of the outside world until you are sure he knows how to look after himself—otherwise you will have him back in coma within a week.

Injectiions.—First he must learn how to use a syringe and how to fill it with insulin. Show him how to withdraw the plunger before sticking the needle into the bottle and to inject air before trying to suck up the insulin. If the needle is short and the bottle half empty turn the bottle upside down. (It is extraordinary how long people will go on sucking up air owing to neglect of these precautions.)

If he is having a combined injection of Z.P.I. + soluble insulin the soluble insulin must be drawn up into the syringe first.

Next you must show him how to sterilise the skin and give the injection. The best sites for self-injection are the front and sides of the thigh, and the abdomen; a fresh site should be used daily if possible.

See that he has a *sharp* needle, knows where to get fresh supplies and how to take care of the syringe.

Urine Testing.—Let him test specimens with and without sugar and acetone, so that he knows what to look out for. Explain that a trace of sugar is less serious than the presence of acetone which should *at once* be reported to the doctor. Be sure he knows how often and at what times he has to test the urine (this will depend on the severity of the disease).

Diet.—This may need more explanation than anything else. It is helpful to have a talk with the wife if the patient is a man. The patient will have a diet chart; see that he knows how to use it.

Insulin Overdosage.—Be sure he knows what are the early symptoms of an overdose of insulin and what he can do to relieve them (see below). The diabetic should always carry sugar, which he can take on these occasions.

There are few diseases in which the results of treatment depend so largely upon the patient's good sense and co-operation. Sensible patients quickly learn how to master their disease so that they can live satisfactory, active lives. Even children generally learn to give their own injections. An intelligent person also learns to adjust his insulin dosage to his needs, and gives himself an extra large dose before a City banquet and a smaller one before a hard game of tennis.

Just occasionally one comes across a patient who is either stupid or stubborn, who *will* not stick to any routine which can be devised. I knew one such patient who was admitted to hospital in coma or

near-coma seven times in one year. It is difficult to know what to do with such people; luckily they are very rare.

Complications of Diabetes

(1) *Insulin Overdosage*.^{*}—This is a complication of treatment rather than of the disease itself, but it is one of the commonest emergencies which diabetics and their associates have to manage.

The symptoms of insulin overdosage come on within a few hours of a dose of soluble insulin, but after delayed action insulins they may be delayed for many hours. The patient feels suddenly giddy, he sweats and trembles and may feel either hungry or sick. These symptoms he should recognize himself, and should immediately eat a few lumps of sugar or some biscuits, which will usually restore him. Later on he should report to his doctor, for if he has many such attacks he is probably getting too little food or too much insulin, and his treatment may need alteration.

Severe attacks of insulin overdosage may be due to a mistake in giving the injection or to a missed meal. In such cases the patient starts behaving oddly†; he may stagger about, talking nonsense, which may lead to arrest for being drunk and disorderly, or he may have a fit or fall down unconscious. If you examine such a patient you will find him drenched with sweat and rather stiff to move; his pulse will probably be normal or only slightly raised.

Insulin overdosage clears so much sugar from the blood that there is not enough left to keep the brain properly supplied; hence the queer mental symptoms, fits and unconsciousness.

Treatment.—First and foremost let us be sure of the diagnosis—I have seen two diabetics killed by huge doses of insulin being given when they were already in insulin coma. In one case the doctor, in the other the patient's mother, wrongly diagnosed diabetic coma and gave the fatal injection.

After an overdose of insulin there is too little sugar in the blood. So we must get as much sugar into the patient as we can, as quickly as we can, in whatever way we can.

If the patient can swallow, give him a drink of water, tea or whatever is handy, in which are dissolved two or three tablespoonfuls of sugar (glucose if available).

* The terms "hypoglycæmia" (low blood sugar) and "hyperglycæmia" (high blood sugar) are best avoided, because they sound so alike that mistakes are often made.

† For instance, one man always used to eat daffodils out of a vase. Another very mild little man used to run amok, and rush into a nearby women's ward, knocking down nurses, porters, or doctors who stood in his way.

If he is unconscious, an intravenous injection of glucose is the best way to restore him. 20–100 ml. of a 50 per cent. solution is given, after which the patient is likely to sit up and look round the casualty ward with puzzled surprise—a dramatic moment for all concerned.

If sterile concentrated glucose solutions are not available immediately, the patient may be given an injection of adrenaline. This raises the blood sugar a little (adrenaline liberates glucose from the liver) and the patient, if not too deeply unconscious, may revive enough to drink his sugar solution.

Glucose may also be given by stomach tube, but intravenous glucose acts far more quickly and certainly.

(2) *Diabetic Coma*.—In pre-insulin days most diabetics died in coma. Nowadays coma may occur in patients with acute, severe, undiagnosed diabetes, in those who have neglected treatment or in those who have developed an acute infection. Any infection—pulmonary tuberculosis, boils or influenza—plays havoc with the body's carbohydrate metabolism and may send the most stable diabetic into coma in a day or two.

Diabetic coma develops more gradually than insulin coma—often over a period of days. The patient suffers from abdominal pain, constipation and vomiting. He breathes quickly and deeply, his breath reeks of acetone, his tongue is dry and brown, his pulse rapid and feeble, and he passes masses of urine which is loaded with sugar and acetone. Later on he becomes confused, semi-conscious, and finally, in severe cases, unconscious, when his *dry skin, parched tongue, sighing respiration* and the *smell of acetone* help to distinguish his condition from that of the man with insulin coma, who is *sweaty*, has a *normal pulse and respiration* and *does not smell of acetone*. You cannot always depend on the urine to give you the diagnosis, because a man in insulin coma may have a bladderful of sugary urine produced before the insulin took effect. In diabetic coma the urine is usually loaded with sugar and acetone. In severe cases there may be no urine because the patient has lost so much fluid that none is produced.

Diabetic coma is caused by the ketones which accumulate in the blood and poison the brain and other tissues; the *blood sugar* is also very high—up to ten times the normal—but this is not the cause of the symptoms. Some of the symptoms of diabetic coma are caused by acidosis and by *dehydration* and *loss of salt*, for the patient loses quantities of water in the urine and in the moisture-laden breath, and salt is lost in the vomit. The dry tongue and rapid,

feeble pulse are due to the dehydration. The *sighing respiration* is caused by acetone.

Treatment.—The patient in diabetic coma is parched, dehydrated and saturated with sugar and ketones; he needs water and insulin at once and in large quantities. He also needs salt to replace what he has lost in the vomit.

A large dose of *soluble insulin* is injected (sometimes intravenously) as soon as the diagnosis is made—40–100 units or more may be ordered. An *intravenous drip* is then set up, and several pints of normal saline (0.9 per cent) or saline lactate are run in rapidly to make good the body's loss of water and salt. As the patient improves *glucose* can be given as well—e.g. 5 per cent. glucose in one-fifth normal saline is run in instead of normal saline.

Potassium is given as soon as the blood sugar is falling satisfactorily. It is usually given intravenously to start with.

Very often the *stomach* needs emptying by means of a gastric tube, for it is often full of a blackish fluid which the comatose patient may vomit; some of the vomit may then be inhaled and cause a fatal pneumonia.

Throughout, treatment is regulated by careful observation of the patient's progress; his condition is likely to change from moment to moment, and every alteration in general condition, respiration, pulse and urine must be noted and reported to the doctor. *Increased breathlessness*, for instance, must be at once reported—it may be a sign of pulmonary œdema, caused by too much intravenous fluid. Encouraging signs are: a clearer mind, quiet breathing, strong pulse, rising blood pressure, moist tongue, and a urine which contains salt, no acetone and not much sugar. A specimen of urine—obtained, if necessary, by catheterisation—should be tested hourly for as long as the patient is having a drip.

Insulin injections are given hourly or two hourly, the dose being regulated by the results of the urine and blood sugar tests. The drip can be discontinued when the patient is fully conscious and has no nausea. He is then given small quantities of fluids to drink, with enough sugar to make up the required amount of carbohydrate. Over the next few days he gradually returns to his usual diet and insulin dosage. In the meantime the cause of the coma must be sought. If this is an acute infection it will require treatment.

(3) *Other Complications of Diabetes.*—The diabetic of many years' standing nearly always suffers from one or other of the following complications: arterial degeneration, polyneuritis, retinitis, cataract or kidney disorder (Kimmelstiel-Wilson disease). The arterial de-

generation may affect the *coronary arteries*, resulting in coronary occlusion and heart failure, or the *cerebral arteries*, causing a stroke, or the *leg arteries*, with gangrene of the toes. Diabetics are more susceptible to all types of infection than non-diabetics—particularly



Fig 65 Gangrene of the toe in a diabetic (Dr Avery Jones's case)

pulmonary tuberculosis. For this reason chest X-rays are always taken on diagnosis, and at intervals after. *Pyelonephritis* is also very common in diabetics. Diabetic women are apt to have still-born babies. Curiously enough, a woman who *later* develops diabetes is liable to give birth to large babies. Giving birth to a series of ten

or eleven pound babies, is a sign that diabetes is likely to develop later—often many years later.

There is evidence that conscientious diabetics, though far from immune, develop fewer severe complications than those who ignore their diet sheets, and whose urine is regularly loaded with sugar and often with acetone. Nurses must urge on their diabetic patients the importance of careful dieting and regular supervision. It is very hard for young people to accept so many restrictions, yet the results of irregular treatment may be the loss of a leg or of eyesight twenty years later.

The Outlook for the Diabetic

There is no cure for diabetes. This means that no diabetic can ever look forward to a time when he can eat exactly what he likes and when he likes, and most cases who need insulin will need it all their lives. Some young diabetics need more and more as time goes on.

Diabetics on average die younger than normal people, and, as we have seen, they are more apt to develop various illnesses than the rest of us. But a sensible diabetic, properly treated, can enjoy a full, active life, and often even his friends are unaware of his condition. Only the most strenuous jobs and those involving irregular hours and much travelling (because of mealtime difficulties) are unsuitable. So this once fatal disease is now a handicap rather than a tragedy, and every effort should be made to encourage diabetics to behave like normal, active citizens, rather than chronic invalids.

Case Histories

(1) *Mrs. T., aged 79*, was seized with an attack of pain behind the breastbone and felt sick and giddy. Next day she had a temperature of 100, auricular fibrillation, a low blood pressure, and her urine was loaded with sugar and acetone. A diagnosis of coronary thrombosis and diabetes was made. We tried to put her on a proper diet and insulin, but she refused her injections and every other kind of treatment, and died in coma in less than a week.

This woman's sister had recently died of diabetes at the age of 80. (Diabetes often runs in families.) The rapidly fatal course of the disease must have been distressingly familiar in pre-insulin days.

(2) *Mr. A., aged 42*, suffered from sciatica. One day he fell down and fractured his femur. He was taken to hospital, and a routine test showed that his urine was loaded with sugar. He turned out to be a moderately severe diabetic. While his leg was mending, he was treated by means of a diet containing 150 gm. glucose a day, with 40 units of Z.P.I. every morning. His sciatica, which was a diabetic neuritis, cleared up as if by magic, and he is now fit and working.

OBESITY AND ANOREXIA NERVOSA

THIS chapter deals with disorders of appetite—with people who eat too much and become fat, and a much less common disorder in which the person eats so little that she becomes a walking skeleton.

Obesity

Obesity is both unattractive and unhealthy. Everyone has noticed that fat people are apt to be breathless and to lack agility. This is partly because of the extra weight carried (imagine carrying around a parcel weighing 20 lb. for the rest of your life). But obesity also causes breathlessness because the diaphragm is pushed up by accumulations of fat inside the abdomen (e.g. in the fold of peritoneum known as the *great omentum*). This makes it more difficult for the lungs to expand; masses of fat round the chest make respiratory movements laborious. (See Fig. 66.) The *heart* may be infiltrated with fat, so that it becomes incapable of responding to extra demands.

Many diseases are both commoner and more serious in the obese. Coronary artery disease causes a much higher death rate among the fat than the thin. Diabetics over the age of 40 are usually obese; sometimes, when the patient regains normal weight as a result of dieting, sugar disappears from the urine. The diabetic death rate among the obese is four times as great as among those of normal weight. Fat people more often have gall bladder disease than thin. The extra wear and tear on the joints of the spine, hips and knees often induces osteoarthritis; rheumatoid arthritis, although not caused by obesity, is much more disabling in the obese. Fat women suffer more obstetric complications than thin, and are more likely to have still-born babies. Fat patients are poor surgical risks, have less resistance to infection and are more liable to develop bedsores.

This formidable list of the dangers of obesity is reflected in the general mortality rates, which are as much as 56 per cent. above the average in the very fat (50 lb. overweight).

Causes of Obesity

Obesity is caused by *eating too much for one's needs*. A fat person has always, *at some period of his life*, eaten more food than his energy requirements demand. The excess need not be very great—an extra



Fig 66 (a) Mrs J. aged 28 Weight 9 stone

Effects of Obesity

Fig 67 (b) Mrs J. aged 38 Weight 11 stone 10 lb Note that the diaphragm has been pushed up. The lungs are compressed and the heart lies more transversely

half an ounce of fat a day, or its equivalent, causes a weight gain of a pound a month—nearly two stone in two years.

Obesity is *not* caused by increased absorption from the gut, or by a low B.M.R. Although weight is often gained in myxædema, Cushing's syndrome and other endocrine disorders, *in the vast majority of cases of obesity the ductless glands are normal.*

Why do people eat more than they need? Eating good food is pleasant, and over-indulgence easy. A man of fifty is seldom as active as he was at twenty, yet the chauffeur-driven executive continues to eat as much food as he was accustomed to do as an athletic student. Similarly, a patient with a fractured femur or pulmonary tuberculosis often gains weight—he goes on eating his normal quantities of food although, while at rest in bed, he needs much less.

Overeating, like drinking, can be an attempt to compensate for unhappiness and frustration. The fat person is not always jolly and sociable—he may be lonely and miserable.

People who stop smoking often put on weight. This is partly because their appetites improve, partly because they are apt to suck sweets or nibble biscuits in an attempt to overcome the craving for tobacco.

A fat person, advised to diet, is apt to say plaintively—"I hardly eat a thing." Such a person usually eats—or drinks—more than he realises, often in the form of odd snacks between meals. A woman may drink numerous cups of sweet tea; it is easy to get through $\frac{1}{2}$ lb. of sugar (880 calories) a week in tea. A pint of beer a day (260 calories) is an appreciable addition to an already adequate diet. However, a fat person is not always a big eater; all we can say with certainty is that *at some period* he must have eaten more than he needed.

The other plaintive cry of the obese is "But look how much so-and-so eats and manages to stay thin." Here again, all we can say is that so-and-so is not eating too much *for his needs*. The lean and hungry type is often very active, and uses up more energy than his fat lethargic neighbour. However, it is something of a mystery why some people have a tendency to get fat and others do not. Those who do not are lucky rather than virtuous.

Treatment of Obesity

Obesity impairs health and shortens life and is well worth taking seriously as a medical problem. In those with diseases such as chronic bronchitis, coronary artery disease or severe hypertension obesity greatly increases the mortality, and weight reduction is often

the most important part of the treatment. It is equally important in diabetes, and in arthritis and similarly disabling conditions.

Obesity is treated by means of a *reducing kiet*. The diet should have a calorie value of, say, half the individual's normal energy requirements—for a woman, about 1,250 calories. Body fat is then used up to supply the calorie deficit, and the person gets thinner. *Strict dieting invariably results in weight loss.* Those who do not lose weight are not keeping to their diets strictly enough.

Reducing Diets.—Weight will be lost if the person drastically cuts down his intake of *all* forms of food. Usually, however, the *carbohydrates*—sugar and starch—are reduced most severely, fats less and protein least of all. This is because carbohydrates are the least valuable part of the diet—sugar, for instance, only supplies energy but no minerals or vitamins, and no bulk to satisfy hunger. Fats give a feeling of satiety (because they diminish gastric secretion and movement) and also supply vitamins A and D. Proteins are important as body builders; also, a protein meal temporarily increases energy consumption, which is useful in weight reduction. Green vegetables, apples, tomatoes, etc. supply bulk to the diet, as well as iron and vitamins; they can be eaten in large quantities because their caloric value is so low.

Those on reducing diets should remember that *all* breads, plain biscuits and similar foods are equally fattening, in spite of beguiling advertisements which state that certain breads, or rye biscuits, are “slimming”. The only exceptions are certain starch-reduced rolls, which have a very low calorie value. Unfortunately they taste like cotton-wool. The following reducing diet is given only as an example; possible variations are numerous.

Calories

Breakfast:

1 egg	80
1 small rasher lean bacon	180
1 oz. bread (1½ slices)	70
1 oz. butter	65
	<hr/>
	395

Lunch:

Clear soup, "Bovril", "Marmite" etc.	0
2 small slices (3 oz.) lean meat	210
1 medium potato (2 oz.)	50
Cabbage, 4 oz.	20
Orange or apple	40
	<hr/>
	320
	<hr/>

Supper:

Helping (4 oz.) of steamed fish	95
1 medium potato (2 oz.)	50
Cauliflower (4 oz.)	20
1 oz. bread	70
1 oz. cheese	120
Butter $\frac{1}{4}$ oz.	65
Helping of stewed apple sweetened with saccharine	50
	<hr/>
	470
	<hr/>

Day's ration of milk, $\frac{1}{3}$ pint (in tea, coffee etc.)	135
	<hr/>

Total = 1,320

Appetite Reducers.—Dexamphetamine sulphate is a drug which reduces the appetite and helps the would-be slim to stick to the diet. It is often prescribed in such cases. *It does not of itself cause any loss of weight.*

Exercise helps to reduce weight *as long as the diet is kept constant or reduced*. Violent exercise often increases the appetite and defeats its own ends. No exercises are known which will remove fat from one part of the body more than another—e.g. from the hips, legs or bust.

Weight-reducing calls for great self control and perseverance. Nurses can help their obese patients by encouraging the discouraged, strengthening the faint hearted and explaining the *medical* importance of a reasonable figure.

Underweight; Loss of Weight; Anorexia Nervosa

Most people are much more afraid of being too fat than too thin, and they are quite right—obesity is harmful, thinness, of itself, is not. Many healthy, active people remain thin throughout their lives, from childhood onwards. Remember this when dealing with the mothers of thin children, particularly thin infants and toddlers. These mothers compare their children's weight with a table of average values, and worry if they are "underweight". An *average* weight is the average of the weight of numerous healthy individuals of that age and/or height. Obviously some of these will be above, others below the average in weight. The thin child is often perfectly healthy—sometimes unusually active—yet he is coaxed and forced to eat more than he wants, till behaviour problems ensue.

To remain thin when plenty of good food is available and the appetite is normal is not a sign of ill health. Pronounced *loss of weight*, however, is of course a symptom of many diseases—infections such as tuberculosis, malignant tumours, severe diabetes, endocrine disorders such as thyrotoxicosis and Simmonds' disease. Loss of weight is often the result of worry or emotional stress—examinations, falling in love, financial worries may all induce weight loss, mainly because the worried person loses her appetite. (As we have seen, however, emotional difficulties *can* lead to obesity.) Women are much more liable to lose weight at times of emotional stress than men. An extreme form of this state of affairs is found in anorexia nervosa (nervous loss of appetite).

Anorexia Nervosa

The patient is nearly always a young woman with emotional difficulties. She may always have been unstable, and there is often a family history of neurosis. The loss of appetite may have started during an unhappy love affair. Sometimes a girl embarks on an overdrastic course of "slimming", and ends by losing her appetite so completely that she refuses food even when seriously emaciated. Nearly always there is considerable friction between mother and daughter—the refusal of food may be the cause or the result of this friction, or a mixture of the two.

The patient with anorexia nervosa finally becomes so emaciated as to resemble a walking skeleton. The trunk and limbs are often covered with soft downy hair. Menstruation usually ceases. The pulse rate is low. The emaciation is so extreme that it is difficult to believe that there is no foundation of physical disease; *Simmonds'*

disease is often suspected. The patient is often restless and surprisingly active; she is moody, unhappy and intensely resentful of attempts to make her eat—especially of her mother's.

Treatment.—Admission to hospital or nursing home is essential; this is an occasion when home influences are usually disastrous. The patient is encouraged to eat; vitamin supplements can be given by injection. A cheerful firmness is required; scolding and nagging must be avoided. *Psychotherapy* is often indicated.

Prognosis.—*Anorexia nervosa may be fatal*, the patient literally starving herself to death. Many patients recover, however, and may regain both mental and physical health.

SKIN DISEASES

DISEASES of the skin are among the commonest plagues of mankind. Though seldom incapacitating and hardly ever fatal they cause an immense amount of distress to their victims, due to itching, disfigurement or both. As a rule, only the severest cases are admitted to the ward, but hundreds are treated daily in doctors' surgeries and out-patient clinics.

The management and treatment of skin diseases is difficult for many reasons. The patient often thinks that there is something shameful and unclean about a skin disorder and may make his rash much worse by trying to treat it himself, frequently with strong disinfectants, before consulting his doctor; in all but the worst cases he has to undertake dressings himself and success or failure may depend upon his understanding how to do this correctly; he often cannot sufficiently protect his damaged skin from everyday hazards in the house or at work—this especially applies to the busy housewife with young children; he may be disheartened by apparent lack of progress so that he either neglects treatment or is tempted to change it; he may not be able, particularly if he is of nervous disposition, to avoid rubbing and scratching the inflamed skin which in turn further inflames it, leading to severe irritation, loss of sleep and considerable mental distress.

It is therefore vitally important for the nurse to adopt a calm, cheerful and optimistic attitude and she can do a great deal to reassure the anxious patient that, except in a few conditions like impetigo and scabies, the skin disorder is neither infectious nor contagious and that it has no relation to uncleanliness either of body or of mind. She should know how to apply lotions, pastes and other skin dressings correctly and should school herself to be patient with the elderly and less intelligent.

Every kind of disease may attack the skin—inflammation of various sorts, tuberculosis, syphilis, cancer and so on. In addition there are a number of skin diseases of unknown causation and others which are included in the allergic group. On the whole, those skin diseases which are caused by infection are much easier to cure than these more obscure varieties. There are some 400 skin diseases altogether, but only a few of the commonest will be described here.

(1) Scabies

Let us begin with scabies, one of the commonest of skin diseases and one of the easiest to cure. Patients who are horrified when they are told that they have what is usually regarded as a very disgraceful complaint should be encouraged by the information that they can probably be cured in 48 hours, whereas, had they developed the more aristocratic eczema, their disease might well have defeated all treatment.

Scabies, or the itch, is an infectious condition caused by a tiny insect known as the itch mite or *acarus*. The female of the species burrows into the horny layers of the skin, laying her eggs as she goes. Eventually she dies, but the eggs hatch out into larvæ which emerge and then take refuge in neighbouring hair follicles. These larvæ develop into adult itch mites after various changes have taken place. Mating of male and female *acari* takes place on the skin, and the impregnated female burrows into the horny layers, thus completing the cycle, which takes about a fortnight.

The *acarus* is a slow mover in its journeying across the skin, and, as a rule, scabies is only passed on between people who are in close and prolonged contact. Symptoms may not appear for several weeks after infection, but the patient is infectious to others during this period.

The *burrows* made by the female are absolutely characteristic and make the diagnosis of scabies certain whenever they are found. The mother *acarus* herself may be extracted from the end of the burrow and examined under the microscope. Burrows appear as greyish, irregular lines $\frac{1}{4}$ – $\frac{1}{2}$ inch long. They are especially common on the front of the wrist, inner side of the hand, between the fingers, on the anterior fold of the axilla and on the buttocks, genitalia and inner surface of the feet. When the body is once sensitised to the presence of the *acari*—which may take a month or more—a rash develops consisting of raised, pinkish, intensely irritating spots, distributed around the burrows and on the abdomen, buttocks and thighs. Itching is so severe, especially when the patient is warm in bed, that sleep may be prevented. The victim often says “I’m just tearing myself to pieces,” and scratch marks are usually seen in addition to the spots and burrows.

The *treatment* of scabies is very simple. It consists of killing the itch mites and their brood by the application of benzyl benzoate emulsion; other preparations include sulphur ointment, Balsam of Peru and zinc cream and mesulphen and one of these may be used should benzyl benzoate fail.

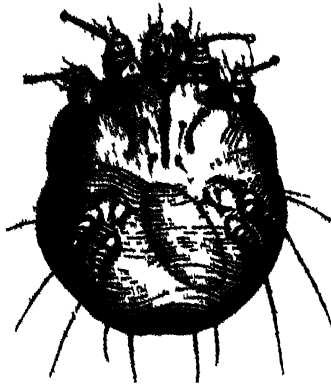


Fig 68 Female Itch Mite (*Acarus Scabei*) magnified
100 times
(From British Museum Economic Series No 6 Stanley
Hurst, 1917)

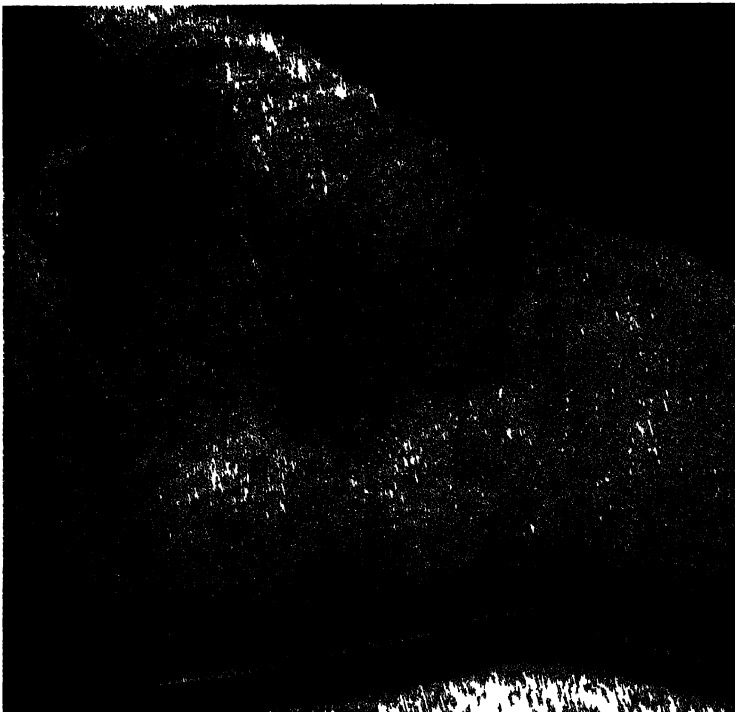


Fig 69 Scabies Burrows on the palm of the hand.

The patient takes a hot bath last thing at night and soaps himself all over with a flannel. After drying, he applies benzyl benzoate all over with a paint brush or old-fashioned shaving-brush. The emulsion is applied to every inch of the skin from neck downwards—not just to the rash—and is worked well into the skin, especially the folds. A second coat is applied when the first is dry. The next night the patient has another bath, puts on clean pyjamas and gets into a clean bed. Next morning he puts on clean underclothes and can usually consider himself cured, though a second treatment next night or a week later is sometimes advised. Linen and underclothes should be laundered, and blankets and outer clothes may be ironed with a hot iron; steam disinfection of bedding is no longer thought necessary.

Any bedfellows the patient may have should be treated at the same time, as they are certain to be infected, and, if possible, the whole family should be treated too. Benzyl benzoate does no harm and it may cure an early, unrecognised case.

Patients should be warned that itching often persists for several days after the treatment of scabies, even though the infection has been eradicated.

(2) Pediculosis

Every nurse should know how to treat scabies and pediculosis (infestation with lice) because patients who are under her care for some other disease may be infested and in need of treatment. Public Health nurses often deal with scores of cases a week, and become very expert in diagnosis and treatment.

The louse is a parasitic insect whose home is on the human body and whose food consists of human blood. Lice lay their eggs in the hair or clothes of their unwilling hosts. *Head lice* attach their eggs to individual hairs with a special "cement"; these eggs are called *nits*. *Body lice* lay their eggs in the clothes; they are common only among the homeless and destitute. *Pubic lice* live among the pubic and axillary hairs, to which they attach their eggs; *pediculosis pubis* is generally passed on during sexual intercourse. In this chapter only the head infestation (*pediculosis capitis*) will be described.

In a really bad case of *pediculosis capitis* the lice themselves are seen as tiny, greyish insects about $\frac{1}{8}$ inch long, crawling about among the hairs; nits alone are found in the milder cases. Small boys and girls often have nitty heads, but after the age of five pediculosis becomes less and less common among boys, whereas older girls are

very frequently infested, especially among town dwellers. (About 50 per cent. of girls under the age of 14, and about 25 per cent. of older girls in industrial towns are affected.) Women are more often affected than men because long hair is harder to keep clean than short. Long hair and elaborate curls favour the louse. Many an elegant coiffure is hardly touched with a comb, let alone a brush, in between rare visits to the hair-dresser, and the mother louse is left to lay her eggs in peace.

Louse bites cause itching and scratching and, in bad cases, the scratch marks become infected, causing septic sores and impetigo of the scalp and enlargement of the glands of the neck. Always search for nits and lice on a patient who has clusters of swollen glands around the back of the neck.

Diagnosis of pediculosis capitis.—

It is generally the nurse's job to diagnose and treat the lousy head. *Nits* are tiny, oval, greyish structures about the size of a pin's head, attached to the hairs, most commonly near the roots. A nit can be run off the hair, but it cannot

be pulled off in any other way, which distinguishes it from a speck of scurf. When searching for nits, have the patient sitting in a chair facing the light, if possible, and stand behind her. Comb out the hair, preferably with the patient's own comb, and turn it all over to one side of the head. Look for nits along the roots of the hairs, especially near the ears. Now make a parting from back to front one inch further inwards. Examine the hairs and their roots along both sides of the parting, and continue in this way, making and examining partings at one-inch intervals right across the scalp. This routine makes it very unlikely that even a sprinkling of nits will escape detection.

Treatment.—The best louse and nit killers are lethane and D.D.T. With lethane, use a dessertspoonful of the emulsion per head, and drop it along each parting, made as described above, using a fountain pen filler or medicine dropper. The lotion is rubbed well into the

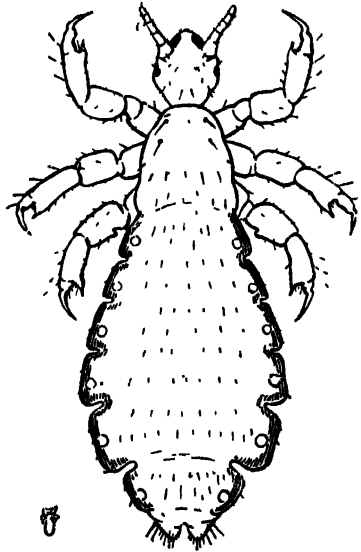


Fig. 70. Female Head Louse, actual size (left) and magnified twenty times (right).
(From British Museum Economic Series No. 2
B. F. Cummings, 1915.)

scalp, which must not be washed for 8-10 days. After the shampoo the head is combed with a nit-comb dipped in vinegar, which removes the dead nits. Ideally the head should be inspected at weekly intervals for several weeks, as it is easy to leave a few nits behind which later hatch out and re-infest the whole scalp.

D.D.T. emulsion is a very efficient de-lousing preparation. The emulsion, which contains 2 per cent. D.D.T. and 5 per cent. naphtha, is worked into the scalp with a two-inch brush; the hair is washed overnight and nit-combed next morning.

(3) Impetigo

This is a very common condition among children, causing crusted sores, especially around the mouth and chin; it is not nearly so common among adults. It is contagious and is caused by streptococci

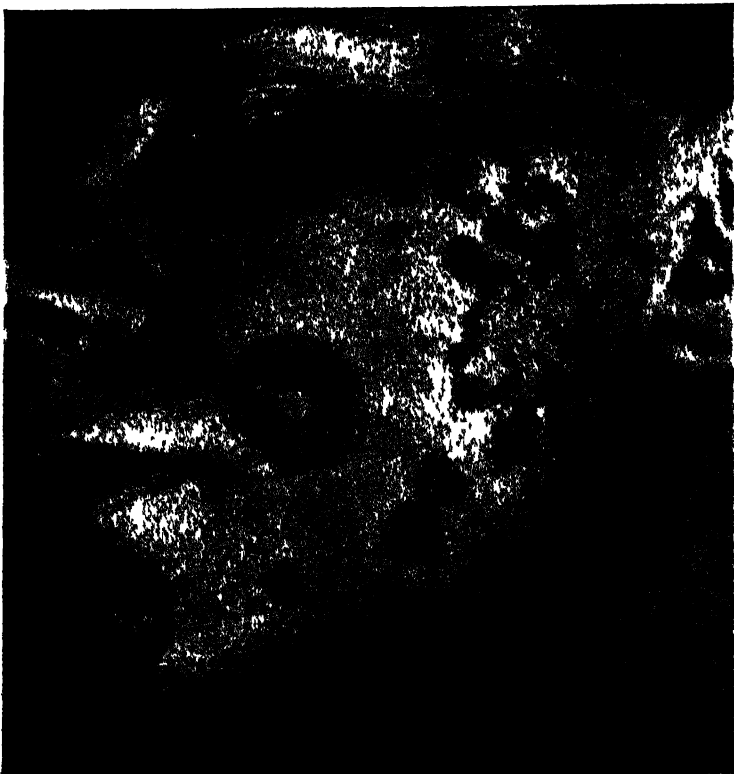


Fig. 71. Severe Impetigo of the Face.
(From A. C. Roxburgh's "Common Skin Diseases." H. K. Lewis.)

and staphylococci, which attack the superficial layers of the skin after entering through some small scratch or graze. Weeping or purulent sores are left behind when the scabs are removed.

Treatment.—In severe cases treatment is best carried out in bed. The first step is to remove the crusts, which may be done with a starch poultice or by mopping with warm olive oil. The crusts once off many applications can be used to cure the underlying infection—e.g. Alibour lotion (copper and zinc sulphate), gentian violet (1 per cent.) in calamine lotion (effective but messy). In resistant cases an ointment containing one of the tetracyclines is often prescribed and is generally effective. (Penicillin applications are not usually recommended as they so often cause sensitivity rashes.) The chosen application is used two or three times a day after removing the crusts until the skin is clear, or until there is no further improvement. If one treatment fails, try another.

(4) Acne

The symptoms of acne need no description, for they may be studied on many a face among one's colleagues and friends as well as among patients. The rash of acne consists of blackheads (comedos), whiteheads and septic spots, occurring on the face, and on the back, shoulders, and chest in severe cases. It is common among adolescents and young adults, and is often a source of miserable embarrassment to its victims. The spots represent sebaceous glands—those glands which produce the natural grease of the skin—which have become clogged and distended by excessive sebum (grease), bacteria and dead cells. The accumulated contents of these distended glands may be infected by pus-forming organisms, such as staphylococci, and septic spots or *pustules* may result which leave ugly pits and scars behind as they heal. The contents of a whitehead consist almost entirely of sebum. Acne afflicts those who have over-greasy skins. It is not infectious. In women acne often becomes worse before the periods.

Every effort should be made to cure acne, which should never be dismissed with a shrug of the shoulders. First of all, these greasy skins need frequent washing in plenty of hot water and soap—which many girls never use on the face. No cold cream or greasy powder base must be used. After a good wash followed by a brisk rub with a towel the largest blackheads should be evacuated. This should *not* be done with the finger nails, which tend to cause scarring. There is a special instrument for the purpose known as a comedo extractor, but anything with a hole in it large enough to admit a

pin will do. The nozzle of a syringe will sometimes serve the purpose. By pressing over the spot the blackhead is squeezed out through the hole. If large pustules are present they may need incision.

After removing a batch of blackheads a sulphur lotion is applied. Such lotions are both irritating and antiseptic, and they tend to cause peeling of the skin. Calamine lotion with added glycerine and sulphur gr. 5-10 to the ounce may be used or a lotion containing sulphurated potash. The lotion is dabbed on, leaving, when dry, a whitish powder which must be rubbed in. After a few days the skin begins to peel. The lotion may be used twice a day for a fortnight unless there is too severe a reaction. Where there are many blackheads or hard papules a paste containing resorcin and sulphur may be used instead of the lotion.

Very obstinate cases may resist all lotions, and ultra-violet light or even X-ray treatment may be successfully used in such cases. Even after the rash has been successfully cleared relapses are very frequent.

Acne is said to be made worse by too starchy a diet and by too many chocolates. Plenty of fruit and vegetables should be eaten by acne sufferers rather than cakes and sweets. Fat patients should keep to a reducing diet, and thyroid extracts may prove helpful. In women, small doses of œstrogens may be given (e.g. Hexœstrol I mgm. daily during the second half of the menstrual cycle).

(5) Ringworm

Ringworm is an infectious skin disease caused by a fungus, which may attack any part of the skin, the nails or the hair. The infection may come from a human or animal source—many domestic and farm animals have ringworm.

Ringworm of the scalp (*tinea capitis*)

This condition is almost confined to childhood, the adult scalp being, for some unknown reason, more or less immune to the ravages of the fungus. The incubation period of *tinea capitis* is from 6-8 days. The infection causes bald patches of greyish, scaly skin to appear about the scalp. These patches are dotted with broken-off hairs.

If untreated, the disease is apt to spread and to persist until puberty. In some cases of animal ringworm (particularly cattle ringworm) a violent inflammatory reaction occurs. The affected part of the scalp is inflamed, boggy and honeycombed with pus. This

condition is called *kerion*;* it is often followed by spontaneous cure, as the infected hairs become surrounded with pus and fall out.

Diagnosis.—If the patient is examined under Wood's light in a darkened room, infected hairs fluoresce with a green light. The diagnosis is clinched if the fungus can be found on microscopic examination of infected hairs.



Fig. 72. Ringworm of the Scalp.
(From A. C. Roxburgh's "Common Skin Diseases." H. K. Lewis.)

Treatment.—*Animal ringworm* of the scalp is less infectious than human ringworm and much easier to cure. The child need not be excluded from school. The head is shaved and Whitfield's ointment (containing benzoic and salicylic acids) rubbed in daily. *Griseofulvin* is given by mouth. This is an antibiotic effective against the ringworm fungus. The adult dose is 250 m.gm. four times a day. Treatment is continued for a month. When the hair begins to grow again the scalp is re-examined under Wood's light to make sure the infection had been eradicated.

Human Ringworm.—The child must usually be excluded from school till cured. The treatment already described is very often effective. *X-ray therapy*, once the standard treatment for ringworm of the scalp, is now rarely necessary. X-ray treatment makes the hair fall out, and the child becomes totally bald within a month. This

* Kerion = honey comb.

usually cures the infection, and when the hair grows again it should be healthy. Examination under Wood's light is a test of cure.

Treatment of Kerion.—Weak compresses of mercuric perchloride are applied to the affected areas, followed by Whitfield's ointment when the acute inflammation has subsided.

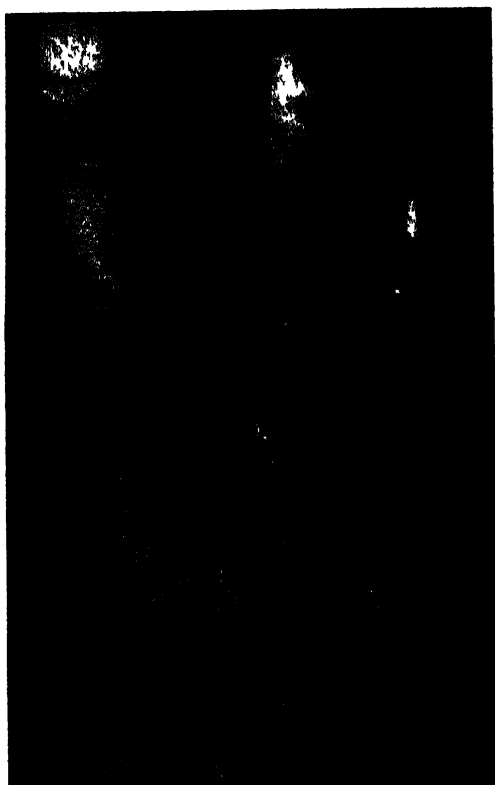


Fig. 73. Ringworm of the Skin of the Hand.
(From A. C. Roxburgh's "Common Skin Diseases", H. K. Lewis)

Ringworm of the beard (*tinea barbæ*, barber's rash) sometimes attacks men's chins. *Treatment* is similar to that of scalp ringworm.

Ringworm of the skin

The *tinea* fungus may plant itself in any part of the skin, where it often causes a ring of tiny blisters and pustules surrounding a red, scaly area—hence the name "ringworm." The infection tends to spread outwards while healing takes place at the centre of the patch.

The itching is intense. This form of ringworm is often caught from domestic animals. The incubation period is from 3–7 days.

Treatment.—These ring-shaped patches are comparatively easy to cure, the fungus being destroyed by various antiseptics—merthiolate cream, Whitfield's ointment, iodine, brilliant green lotion, gentian violet and calamine lotion. Griseofulvin will usually cure this type of ringworm, but is rarely necessary. Any clothes which come into contact with the rash must be boiled or cleaned.

Ringworm of the groins (*tinea cruris*)

Ringworm is particularly apt to attack moist areas such as the groins and axillæ. Large red inflamed patches form, the inflammation and the itching being most intense at the edge of the patch. The condition is highly infectious; it is transmitted by direct contact or by clothing or towels.

Treatment.—The patient should have two pairs of cotton pants which are boiled on alternate days; affected parts must be thoroughly washed daily to remove scales. Mild cases respond to Whitfield's ointment twice daily; if this fails one of the other fungicides already mentioned may be tried. Griseofulvin may be very effective.

Ringworm of the feet

This is the commonest of all forms of ringworm and is one of the commonest of skin diseases, affecting a large proportion of the population at one time or another. The infection is picked up from bath-mats, changing rooms, bathing places and wherever people go barefoot. The first symptom is generally itching between the 4th and 5th toes, where the skin becomes sodden, cracked, moist and inflamed. The clefts between the other toes and sometimes even the rest of the foot may be infected. Itching is intense and infection with pus-forming organisms may supervene. The dead skin eventually peels off from the affected areas. Such a condition is often known as *athlete's foot* (other infections besides ringworm can cause athlete's foot).

Treatment easily checks the infection but permanent cure is very difficult, because each patient constantly re-infects himself from his socks and slippers. Whitfield's ointment or any of the fungus killers already mentioned will practically always clear up the infection for the time being. Dead skin should be removed daily with forceps. Socks and stockings may be disinfected by soaking them for one hour in 1 per cent. thymol in spirit; shoes may be treated by

sealing them up in a biscuit tin for 48 hours with a saucer of formalin, whose vapour kills the fungus.

Ringworm may attack both the finger and toe-nails; in the latter case ingrowing toe-nail may result. Griseofulvin is very promising in the treatment of ringworm of the finger-nails, but usually fails to cure ringworm of the toe-nails. Infected toe-nails should be removed surgically, and the nail-beds treated with fungicides. If—as often happens—the new nail proves to be infected, the nail-bed may have to be excised, so that no new nail can grow.

(6) Allergic Conditions of the Skin

Allergy means an *altered reaction*. In allergic conditions the person reacts abnormally to something which is harmless to normal people, or harmful only in much larger doses. The substance provoking this abnormal reaction is called an *allergen*. Before an allergic reaction can occur the person must become *sensitised* to the allergen. Sensitisation is thought to occur in this way: the allergen (which may be a drug, or certain plant or animal proteins) reaches the skin from the outside or via the blood stream. At first there is no visible effect, but the presence of the allergen excites counter-substances — *antibodies*—which become fixed to the skin cells. The patient is now sensitised, and if the same allergen reaches the skin on some future occasion—even in minute amounts and for a short time—a reaction between allergen and antibody occurs in the skin cells. This reaction is the cause of the rash in allergic skin conditions.

Allergy also plays an important part in the causation of asthma (p. 134) and hay fever (p. 352).

Only certain people are liable to develop allergic conditions; the tendency is probably inborn, and it often runs in families. Different members of the family may suffer from asthma, hay fever, urticaria or eczema. A person may have both eczema and asthma, together or at different times (see below).

All allergic conditions are much influenced by the patient's mental and emotional state. We have seen that this is true of asthma; it is equally true of allergic skin conditions. In these the rash may be a sign of anger, frustration or anxiety, just as blushing is a sign of embarrassment in normal people and "goose-flesh" a sign of fear.

(a) Urticaria (Nettle Rash)

Attacks of urticaria may be precipitated by a great variety of substances. Some people have an attack whenever they eat an aspirin tablet, a strawberry or a shrimp. *Penicillin* by any route may produce

an urticarial reaction in a sensitized subject—particularly local applications of penicillin, such as creams and lotions. A very severe urticarial rash may follow an injection containing *horse serum* (e.g. anti-tetanic serum, diphtheria antitoxin), especially if the patient has had serum in the past; the first injection *sensitises* him to subsequent doses of serum. (In “serum sickness”, as it is called, fever, vomiting and joint pains often occur as well as urticaria.) Allergic subjects—e.g. asthmatics—are particularly liable to have severe serum reactions.

All these foods, drugs and sera contain substances which can act as *allergens*. Many urticarial subjects have attacks for no apparent reason, the rash appearing and disappearing with a strange waywardness. Or they may develop urticaria if they are over-heated, or if they fly into a temper.

The patient may have attacks at irregular intervals throughout his life; sometimes attacks occur almost daily for a period, and then clear up for no apparent reason.

The rash in urticaria consists of red blotches and white wheals, like nettle stings—hence the name (*Urtica* is the Latin name for nettle). These appear with bewildering suddenness, so that large areas of the body may be covered with wheals in less than half an hour. The rash is intensely itchy. It may last for minutes, hours or longer, and may disappear as suddenly as it appeared.

Treatment.—As in the case of asthma we do not know how to remove the tendency to urticaria. If the exciting cause is known—whether it be some food, drug, or contact with plants or animals—it should be avoided. Urticarial subjects generally have skins which are unduly sensitive to sunburn and should avoid over-indulgence in sunbathing.

The individual attack of urticaria should be treated with adrenaline, ephedrine or one of the *anti-histamine drugs* internally and, externally, a cooling lotion to allay itching. Injections of adrenaline (5–10 minims) or the less powerful ephedrine (gr. $\frac{1}{4}$ – $1\frac{1}{2}$ by mouth) cause constriction of the superficial blood vessels which, in this condition, are overdilated, thus allowing serum to ooze out with the formation of wheals. Ephedrine tablets may be taken two or three times a day when attacks are recurrent.

Locally, calamine lotion with added phenol or lead subacetate is a good remedy for the itching. It should be dabbed on to the affected skin and allowed to dry. A sedative, such as phenobarbitone gr. $\frac{1}{2}$ twice daily, with gr. 1 at night, may be necessary when the irritation is intolerable.

The anti-histamine drugs—e.g. promethazine (“phenergan”),

mepyramine ("anthisan")—counteract the effects of *histamine*, which is thought to be the cause of various allergic symptoms such as urticaria. These drugs may be taken regularly, to ward off attacks, or during an attack to cut it short. The disadvantage of such treatment is that it is apt to make the patient sleepy and confused.

(b) Angioneurotic Oedema (Giant Urticaria)

This is a more severe form of urticaria, affecting the subcutaneous tissues. Swellings appear here and there about the body with extraordinary suddenness. The swellings may be an inch or two across or may cover large areas. The skin over them is often red and hot. An attack seldom lasts longer than a day or two, but recurrences are frequent; often the same part of the body is affected each time. If the swelling affects the tongue or throat the patient's life may be in danger from suffocation.

Treatment is as for urticaria. Injections of adrenaline are indicated in severe cases.

(c) Papular Urticaria (Heat Spots)

This rash is extremely common in infants and children under the age of three. The rash is intensely irritating and affects the trunk more than the limbs. It consists of small raised red spots each surrounded by a pink blotch. Scratching often leads to infection and impetigo is superimposed. Attacks may last days or weeks and often recur. It is possible that the rash, in some cases, is the result of an allergic reaction to insect bites—for instance mites carried by budgerigars. *Treatment* is similar to that of ordinary urticaria.

(d) Eczema and Dermatitis

The rash in these two conditions is identical. In eczema the skin becomes inflamed because it has been *sensitised*, as described above. In dermatitis the inflammation is caused by skin *irritants*. Irritants differ from sensitisers in that they will cause inflammation on first contact with anyone's skin, provided that they act for long enough and are sufficiently concentrated. Almost anything under the sun—including the sun—can sensitise or irritate somebody's skin, but some things are particularly prone to do so—for example, certain *plants*, *chemicals* such as mercury compounds or formaldehyde, or local *anæsthetics* in ointment form. Many *drugs* can sensitise the skin, either when given internally or applied as dressings, notably penicillin and sulphonamides (as we have seen, penicillin can also cause urticaria). Internal allergens can be formed in the body from the

products of tissue breakdown elsewhere in the body. The reaction to sensitizers and irritants is more severe if the skin is *damaged from without* (as by strong sunlight, excessive soap and water, detergents, friction). Disturbances of the *general health*, especially infection and psychological upsets, also exacerbate these skin reactions. The *constitutional nature* of the skin is equally important.

Eczema or dermatitis is thus a skin reaction due to a combination of at least three factors:

- (1) A constitutionally sensitive skin.
- (2) Predisposing factors such as friction, infection, irritants.
- (3) Specific sensitisers (allergens).

The first two are far the most important, and very few cases of eczema are due to one specific cause.

Eczema may occur in old or young, male or female; the patient may have a single attack or repeated attacks may occur throughout life. The patient's near relations may suffer from eczema or from other allergic conditions such as asthma, hay fever or urticaria. The condition is then called *atopic eczema* (atopy = family history of allergy).

Atopic eczema often starts in infancy. *Infantile eczema* usually affects bottle fed babies—often plump, lively and intelligent children. The baby frequently has a dry skin and may be sensitive to milk, eggs, cereals or other foods. The face is most often affected—cheeks, brow and chin—but the rash may involve the limbs too. Itching is severe, and the baby makes the rash worse by scratching and rubbing. Secondary infection is common, and, later, a leathery thickening of the skin known as *lichenification*.

The baby who has infantile eczema may get over his skin trouble in early childhood. Often patches of dry eczema affecting the *flexures* of the knees and elbows persists into later childhood (Besnier's prurigo). Many of these children develop asthma.

The rash in eczema.—The rash may occur anywhere, but exposed parts of the body are usually affected. The first sign of eczema is usually a red patch, which quickly becomes covered with *vesicles*—tiny blisters about the size of a pin's head. The itching is severe, and the tops of the vesicles are often rubbed off, leaving a weeping surface. The serum which pours out dries and forms a crust, and secondary infection is very likely to occur. Later on the rash dries up and the skin becomes thickened and scaly; it is still irritating, and the constant rubbing and scratching prevent healing. At any time a relapse into the acute vesicular stage may occur, due to scratching, emotional upset or unsuitable treatment. The localised eczema may then flare up, or a generalised spread to distant parts of the body may

occur. This is particularly common in eczema of the legs in women with varicose veins, where the nutrition of the skin is poor, and it is therefore much less resistant than normal skin.

Treatment of eczema.—This is difficult owing to our ignorance of the cause of the condition. Various internal remedies have been suggested, but they are not of great value. Alkalis and Vitamin B preparations may help patients with greasy skins. Antibiotics such as tetracyclines are given if there is much infection. Steroids will sup-

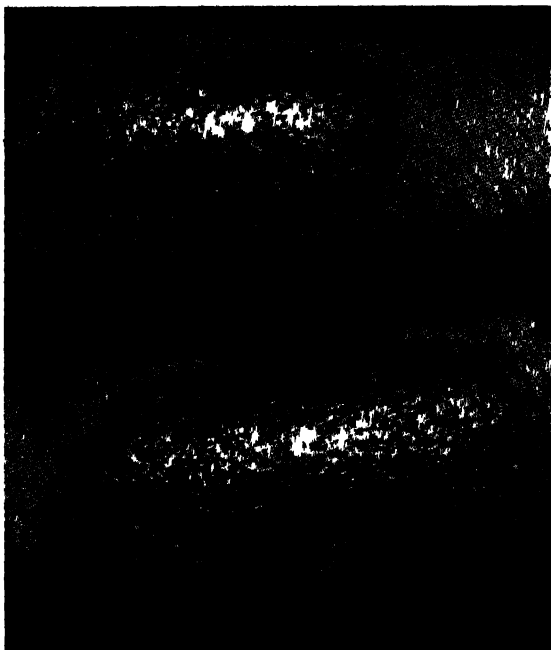


Fig 74. Eczema of the Forearms The rash is in the acute vesicular stage (Dr H G Adamson's Case)

press an attack, but are best avoided as relapse occurs as soon as they are withdrawn, and this disheartens the patient. Sedatives are necessary in severe cases. When the rash is very widespread the patient is best treated in bed; the required dressings are easier to apply and the friction of the clothes is avoided.

In treating the *rash* our aim is to allay irritation and to protect the damaged skin from infection and injury.

First of all, water and soap are bad for eczematous subjects and should be kept away from affected areas if possible. The part should be cleaned up with cold cream and protected with an ointment

when the patient takes a bath. Cold winds, hot sunshine and hot fires should likewise be avoided.

In the acute vesicular stage *calamine lotion* is soothing and protective, and 1-2 per cent. phenol, menthol or chloretone may be added to allay irritation. Calamine lotion dries and leaves a powder, which both cools and protects the skin. In the weeping stage of the rash,



Fig 75 Infantile Eczema

Note that the rash is confined to forehead cheeks and chin—the usual distribution (Dr H G Adamson's Case)

gauze wrung out in lead lotion may be used. The lead salt forms an insoluble precipitate with the oozing serum and this protects the raw area. All dressings in eczema should be as thin and loosely applied as possible to avoid overheating the part and increasing the irritation. *Crusts*, if present, should be dealt with as in impetigo.

Once the acute stage is over oily preparations will help to protect the skin and prevent its becoming too thick and scaly. *Calamine*

liniment (calamine, olive oil and lime water) is useful, and any of the irritation relievers mentioned above can be added. Only in the chronic stage, when the skin is dry, thickened and cracked, may pastes and ointments be used; they keep in heat and moisture too much to be applied in the acute stages. Zinc paste, spread on lint dressings, forms a simple protective covering. Coal-tar ointment, with salicylic acid to remove the over-developed horny layer, may be used when there is much thickening. In very chronic cases X-rays are often used to treat resistant patches.

Local applications of steroids, as lotions or ointment, are extremely valuable for treating small areas of eczema, specially in places where the skin is thin, such as the eyelids, external auditory meatus or anogenital region. An antibiotic such as neomycin may be added to the steroid preparation.

Any one attack of eczema may be cleared up, or may run its course uninfluenced by treatment, but these subjects are always liable to relapses, which cannot be prevented as long as we remain ignorant of the true cause of the condition.

The skin is often affected by the subject's mental state, as is shown by normal reactions such as blushing, turning white with anger or goosefleshy with fright. Attacks both of urticaria and eczema may be precipitated or prolonged by emotional upsets. A calm, cheerful attitude is necessary in those treating such conditions. Patients with skin diseases easily become depressed, and so do their doctors and nurses, owing to the slow progress which is often all that can be expected. In addition to great patience the most scrupulous cleanliness is needed in nursing these patients. If an eczematous rash becomes infected the results may be serious.

The eczematous infant needs special protection against infection, as these fat babies seem to be very prone to contract infantile diarrhoea and other general infections. If they have to be admitted to an infants' ward they should be barrier nursed.

Lotions and pastes may be applied to the face—the usual site of eczema in infants - on a lint mask with holes cut for the eyes, nose and mouth. The best way to stop the scratching which so often prolongs the disease is to stop the itching. This may be achieved by a combination of soothing applications and general sedatives such as chloral or phenobarbitone. It may also be necessary to tie the child's hands to the side of the cot, or to apply cardboard splints to the arms to prevent him from getting at his face. In such cases a sedative should always be given; it is needlessly cruel to tie him down and leave him to wail in an agony of frustrated itching.

Dermatitis

Dermatitis literally means inflammation of the skin, but the term is usually applied only to those cases of superficial inflammation due to known poisons and irritants. The poison may reach the skin from without or via the blood stream. Some drugs, such as gold and arsenic compounds, may cause a very severe form of dermatitis. However, the much commoner forms of dermatitis are those due to external chemical irritants.

In these cases the symptoms are those of eczema and the treatment is the same; the patient must, of course, avoid all contact with the responsible irritant. The rash is often caused by some chemical the patient uses at work; it is then called *trade dermatitis*. If a worker is incapacitated by trade dermatitis he is entitled to compensation under the Workmen's Compensation Act. Hence the word "dermatitis" should never be used to a patient to describe some other type of rash, or he will think he is entitled to compensation.

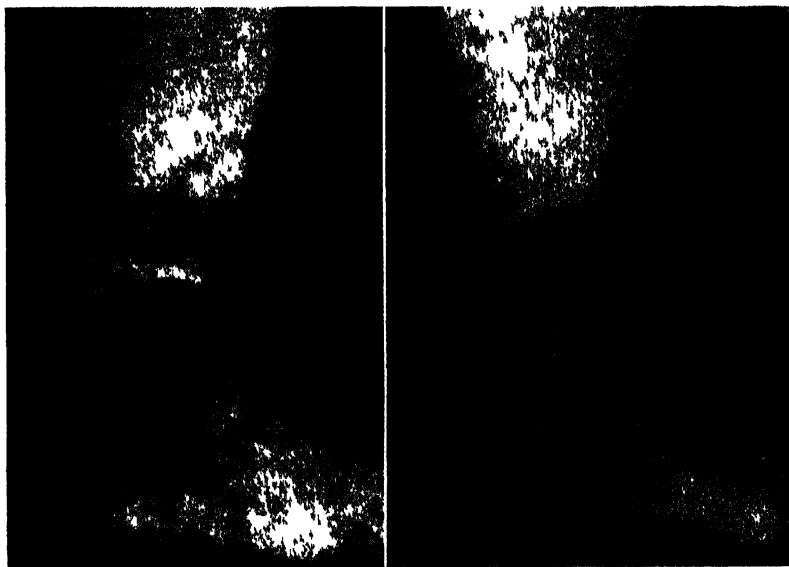


Fig. 76 Varicose Ulcer.
Before and after treatment with stockinette tight bandaging (Dr. Keith Ball's case)

Dermatitis may be caused by a great variety of chemicals, and sometimes it is difficult to be sure which of several substances used by the patient is the cause of his trouble. Soap and detergents may cause dermatitis in housewives; certain chemicals used in breadmaking

may cause *bakers' dermatitis*, and petrol, tar, benzene, cement, chemical dyes and many other substances may also be responsible for skin disease. The rash affects the area chiefly exposed to the irritant; for example, *dishwasher's dermatitis* naturally affects the hands and forearms, whereas *cement dermatitis* may be limited to the feet and ankles, which are attacked as the man tramps through the cement dust in the brickyard.

Measures to prevent industrial dermatitis include the provision of proper washing facilities and the use of barrier creams. Housewives with dermatitis of the hands should use rubber gloves for washing up and washing clothes, or a plentiful supply of barrier cream.

(7) Chronic Leg Ulcer

Chronic leg ulcers are very common, especially among middle-aged and elderly women, and are a cause of much distress and disability. These ulcers are usually found on the inner surface of the lower part of the leg. The edge of the ulcer is hardened, the base purplish and oozing with serum. The ulcer is painful, and may be so large as almost to encircle the leg. The surrounding skin is purplish and scaly, and the whole leg is often swollen. Varicose veins are often present.

Chronic leg ulcers develop because there is some obstacle to the return of venous blood from the leg. On account of this, venous blood stagnates in the foot and leg and the nutrition of the tissues is impaired. Resistance to infection is low, and any slight injury to the skin may lead on to the formation of a chronic ulcer.

Most commonly the trouble originates in a thrombosis of the deep veins of the leg (p. 92); this may have followed childbirth or an operation. Venous thrombosis usually destroys the valves which, in normal veins, prevent backward flow of the blood. *Varicose veins* may also be associated with chronic leg ulcer, but they are rarely the whole cause of the trouble as was once thought.

Treatment.—The most important part of the treatment is to *reduce œdema* and *improve the venous return from the leg*; if we can achieve this the ulcer will often heal. In addition, local treatment of the ulcer itself and of eczematous skin may be necessary.

Swelling subsides, the skin improves and the ulcer usually heals if the patient spends a few weeks in bed, without any other treatment.

When she gets up, the patient must invariably wear an elastic stocking or an elastic supporting bandage, or the ulcer will soon break down again. The bandage should be of elastic web, or if this cannot be tolerated, the lighter but less efficient elastic net may be

used. It should be put on before the patient gets out of bed in the morning, when swelling is at its least and should closely encase the limb from the base of the toes to the knee.

If the patient cannot afford the time to go to bed, an elastic bandage plus local treatment for the skin and the ulcer will be necessary. If the ulcer is discharging copiously, *eusol* and *paraffin emulsion* is a useful antiseptic application. An absorbent dressing is applied, with the elastic bandage over all. *Lassar's paste* may be applied to eczematous skin. When the ulcer is clean and no longer discharging, tulle gras is often used.

When the ulcer is well on the way towards healing, a bandage impregnated with zinc-gelatine paste is often used. The bandage is rolled on wet, and several layers are applied. The paste sets when dry, forming a slightly flexible support; the zinc paste is soothing and protective to the skin. An elastic bandage can be applied over the (dry) zinc paste bandage; the latter can be left on for a week at first—later, for two weeks.

Finally, when the ulcer has healed and the eczema has cleared up, an elastic bandage or elastic stocking can be used without any other application. Some form of support will probably have to be worn *more or less indefinitely* or the ulcer will recur.

Surgical treatment is necessary in some cases; the results are variable.

(8) Psoriasis

The cause of this very common condition is entirely unknown. Psoriasis may develop at any age, but the first attack usually occurs between the ages of 5 and 15. Sometimes a generalised psoriasis rash follows an infection in childhood; this may clear up and never recur. More often patches of psoriasis develop and, though they may clear up, with or without treatment, they tend to recur throughout life. Isolated patches sometimes spread and involve large areas of the skin, including the scalp.

Isolated patches are most often found on the knees or elbows. A patch of psoriasis forms a raised, reddish plaque with a sharply defined edge. It is covered with silvery scales which can be rubbed off, leaving minute red dots exposed. If the palms and soles are involved the skin becomes grossly thickened and cracked. Psoriasis may also involve the nails, which become pitted like a thimble, or thick, rough and breakable.

Treatment.—The *acute generalised psoriasis of children* required only a bland application such as zinc cream, together with salicylates by mouth.

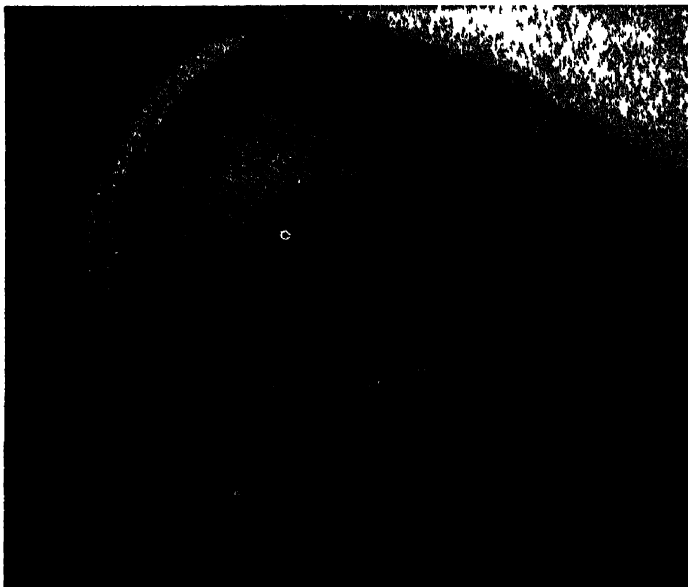


Fig. 77. Patch of Psoriasis on a boy's knee.
(From A. C. Roxburgh's "Common Skin Diseases". H. K. Lewis.)



Fig. 78. Psoriasis of the Finger Nails.
(From A. C. Roxburgh's "Common Skin Diseases". H. K. Lewis.)

In the *spreading psoriasis of adults* zinc cream is also used in the acute stages. Later, a 2 per cent. salicylic acid ointment is used for a few weeks. Later, tar and mercury ointment can be used. By mouth, calciferol (Vitamin D₂) is sometimes prescribed.

For *chronic patches of psoriasis* tar or salicylic acid preparations are also used. *Dithranol* is more effective but can cause severe reactions; it is therefore better to take the patient into hospital. Dithranol paste must be applied to the psoriasis plaques only—not to the normal skin. In hospital *tar baths* and *ultra-violet light* can also be given. A tar bath contains several ounces of liquor picis carb. The patient has two or three tar baths a week. The day after the bath the patient is treated with ultra-violet light.

Very resistant patches of psoriasis usually respond to X-ray treatment.

Prognosis.—Intensive treatment will usually clear up any one attack, but recurrences are common.

(9) Erythema Nodosum

In this conditior tender, deep-red lumps appear on the skin of the legs, especially over the shins; the rash rarely affects the rest of the body. The lumps may be the size of a pea or much bigger. The



Fig. 79. Erythema Nodosum. (Dr. Sakula's case.)

patient is often feverish, with a sore throat and pains in the joints.

We do not know what causes erythema nodosum, but it often follows closely on a first infection with tuberculosis; it appears at about the same time as the Mantoux test becomes positive (p. 109). The tubercle bacillus is not found in the skin lesions. Erythema nodosum may also complicate various streptococcal illnesses.

The *treatment* is that of the primary disease; the patient should be kept in bed in the acute stage. The rash generally disappears within a month. The patient is always investigated for tuberculosis.

(10) Common Tumours of the Skin

Tumours may develop in the skin, as in other parts of the body, and as elsewhere, these tumours may be malignant or benign. Benign tumours of the skin are much commoner than malignant.

Moles (Nævi) are congenital tumours consisting of overgrowths of certain structures in the skin.

Pigmented moles are extremely common; most people have one or two. They may be flat or raised, large or small, are brown in colour and sometimes hairy. The vast majority are best left alone. If removal is essential, because they are noticeable and ugly, or because they are exposed to friction, surgical excision, with or without skin grafting, will be required.

Vascular nævi (hæmangioma) consist of local collections of abnormal blood vessels.

Port wine stain (capillary nævus) is a flat, reddish purple mark, usually found on the face or neck. It can be very disfiguring. There is no entirely satisfactory treatment. Thorium X, a radioactive substance which can be painted on and which just penetrates the top millimetre or so of skin, is often helpful. Several applications may be necessary.

Strawberry mark is another type of blood vessel tumour, known as cavernous hæmangioma. It is raised, bright red in colour, and looks as if it were stuck on to the skin. Other cavernous hæmangiomas are embedded in the skin, to produce a dusky blue swelling with a red top. These nævi tend to enlarge during the early months of life but over the next few years they gradually become flatter, paler and finally disappear without treatment in the vast majority of cases. The mother should be constantly reassured that this will probably occur. If treatment is required, the nævus may be frozen with CO₂ snow for a few seconds; this hastens the natural process, but at the risk of leaving a scar. Very occasionally a hæmangioma near the eye

or mouth enlarges rapidly and interferes with sight or sucking; treatment with small doses of radium may then be necessary. If exposed to friction, as in the napkin area, these *nævi* may ulcerate, but usually heal well if protected with a simple ointment and a dressing. Surprisingly enough, serious bleeding from a *hæmangioma* is very rare.

Seborrhoeic Keratoses.—These are the flat brown excrescences so commonly found on the trunk in elderly people. They can readily be scraped off with a curette, or treated by freezing or cauterization.

Warts (Verrucæ).—These lumps of overgrown skin are the result of a virus infection, so that warts are not tumours in the strict sense. They are very common, especially in children; the hands, knees and feet are most often affected. On the soles of the feet they are called *plantar warts*; these are particularly tiresome as they cause pain on standing or walking. Warts are contagious, but only certain people seem to get them. Their behaviour is quite unpredictable—they may persist for years and recur in spite of vigorous treatment, or they may suddenly disappear by themselves, almost overnight. All kinds of stories are told of warts being cured by wart-charmers, who prescribe weird rituals, such as burying a piece of stolen meat by the light of the full moon. These stories are probably true—it is well known that warts can be cured by hypnosis, which, like the wart-charmers' ritual, is a form of suggestion.

More often, however, warts are treated with a salicylic acid and collodion paint, which is applied daily to the wart and the skin immediately surrounding it. This is continued, if necessary for weeks, until the wart has gradually peeled away. *Plantar warts* may be soaked for ten minutes twice daily in 3 per cent. formalin. After about six weeks the wart dries up and crumbles away, or the remains can be painlessly scraped out. Resistant warts in adults may need burning off with electrocautery under a local anaesthetic, or freezing with CO₂ snow.

Malignant Tumours of the Skin.—These may be found anywhere, but one type (rodent ulcer) is usually found on the face, often near the outer corner of the eye. Many malignant tumours can be successfully treated surgically or with radium or X-rays. *Malignant melanoma* is the exception—this blue-black tumour often spreads widely in the blood stream before the primary tumour can be excised; the mortality is high.

DISEASES OF THE NERVOUS SYSTEM

(1) Stroke

By far the commonest disease of the nervous system is a paralysis caused by the cutting off of the blood supply to part of the brain. Such a paralysis is called a *stroke* or *apoplexy*. It is entitled to pride of place in a book for nurses, not only because it is so common, but because the treatment of stroke is the test of good nursing. Medicine can do little for these patients; their future is in the hands of the nurse.

Apoplectic disorders cause nearly a seventh of all deaths in England and Wales, and are one of the commonest causes of death. Most of the victims are over sixty. It is thought that there are some 700,000 people in this country suffering from a stroke or its after effects.

In apoplexy almost any of the arteries of the brain the *cerebral* arteries—may be affected, so that any of the functions of this all-important organ may be upset. Many varieties of stroke are therefore possible. But it so happens that one particular artery, called the *middle cerebral*, is more often affected than any other, and the common variety of stroke which follows is a *hemiplegia*, or paralysis of one side of the body.

A patient who has just had a severe stroke is often admitted as an emergency. He is unconscious, breathing deeply and noisily; his face is flushed, his pulse full and slow. He may be so deeply unconscious that nothing will disturb him, and he may be undressed and put to bed without offering either resistance or assistance. In less severe cases, although the patient seems to be quite unconscious he resists being disturbed and may groan and make indistinct noises. The arm and leg on one side are usually noticeably limper or more immobile than on the other. The unconscious patient may be incontinent, or retention of urine may occur.

The patient's relatives may be able to describe how the stroke occurred. Perhaps they heard him fall and went into his room to find him unconscious on the floor. Sometimes a stroke comes on gradually, when the patient complains of heaviness and weakness, or of numbness and tingling in the limbs of one side of the body. The weakness increases until the limbs are completely paralysed and

the patient may then gradually become unconscious. Slight strokes do not cause unconsciousness at any time.

After a severe stroke the patient may die within a few hours or days. In non-fatal cases the patient recovers consciousness within a day or so, to find himself paralysed down one side of the body and, perhaps, unable to speak. One side of the face sags and the wrinkles



Fig 80 Stroke

Right sided hemiplegia. The head and eyes are turned away from the paralysed side. The right arm is kept under the bedclothes (a characteristic position among hemiplegic patients) (Dr Richard Asher's case)

seem to be ironed out. The paralysed limbs gradually stiffen, although, while the patient was unconscious, they were quite limp. The leg tends to stiffen with the knee straight and the foot pointing downwards; the arm is held close to the body with the elbow, wrist and fingers bent—a position rather like that of the front paw of a dog begging.

Speech is likely to be upset if the right side of the body of a right-handed person is paralysed. The patient can generally understand what is said to him and can often read; he cannot speak, however, although there may be no signs of paralysis of his tongue and lips. This condition is called *aphasia*.

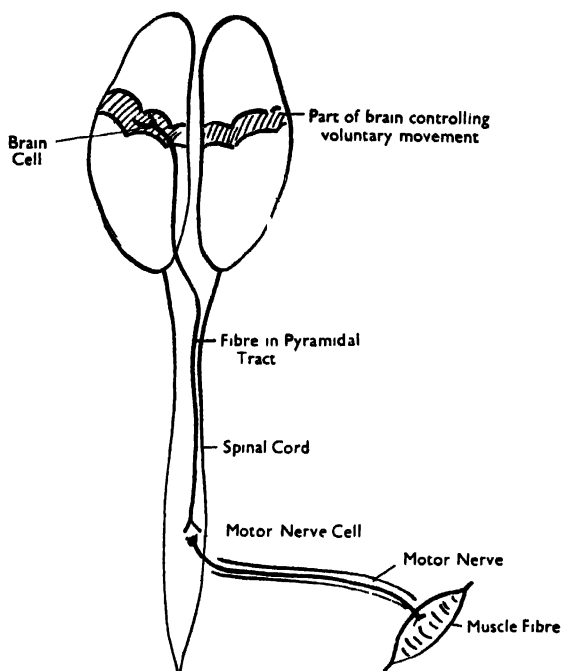


Fig. 81. Diagram to show how the brain and spinal cord control the movement of muscles

A hemiplegic patient often recovers many of his lost powers during the first few days or weeks after the stroke. The face recovers first, then the leg and, lastly, the arm and hand. Fine movements of the hand and fingers, such as are involved in writing or playing an instrument, are the last to return. When he begins to walk the foot which, when paralysed, was held pointing downwards, tends to drag and cannot be lifted clear of the ground unless the patient swings the leg outwards as he walks—you can often recognise a person who has had a stroke by this characteristic way of walking. The speechless patient often begins to talk after a few days, though he may always have difficulty in remembering names thereafter.

The meaning of the symptoms.—All these symptoms are due to the cutting off of the blood supply to those parts of the brain which

control *voluntary movement* and *speech*. Voluntary movements—those movements which are under the control of the will—are controlled by a ridge of tissue running from side to side across the middle of the brain. This ridge, or the nerve pathways (*tracts*) leading from it, is damaged when the stroke occurs. The left half of the brain controls the right side of the body and *vice versa*.

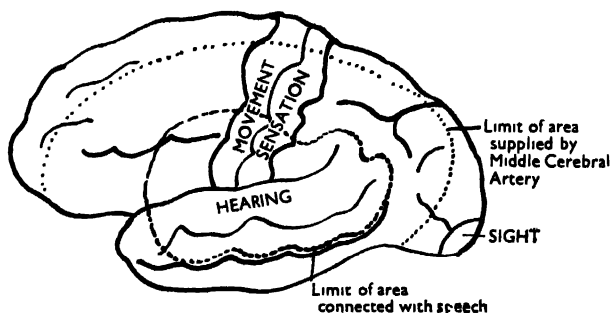


Fig. 82. Diagram of the left side of the brain showing areas concerned with voluntary movement, sensation, hearing, sight and speech.

Speech (in a right-handed person) is controlled by certain parts of the *left* side of the brain. Speech and writing are such closely linked functions that they are generally lost together, and aphasia, when it occurs, is always associated with right-sided hemiplegia in a right-handed person.

What accounts for the stiffness of the paralysed limbs? This stiffness (*spasticity*) comes on because the muscles still retain their own nerves which arise in the spinal cord. These nerves are normally under the control of the brain and, when this control is removed, they tend to overact, so that the muscles are tense and rigid. Similarly, although *voluntary* movement is lost, being controlled by the brain, the *reflexes*, such as the knee jerk, are actually increased in vigour, for they are dependent only upon cells in the spinal cord.

The cause of the stroke.—The cutting off of the blood supply which has such serious results is most often due to the blockage of the artery by *thrombosis*. Blood clots in the artery because its wall is diseased, as may happen in hypertension. The most severe and fatal strokes occur when a diseased artery bursts, allowing the blood to gush out and plough up the brain tissue, so that recovery is practically impossible. *Cerebral hæmorrhage*, as it is called, most often occurs in hypertensive patients, whose cerebral arteries finally become unable to stand up to the high pressure inside them. After a cerebral hæmorrhage the blood may work through to the

surface of the brain and mix with the *cerebro-spinal fluid* which bathes the brain and spinal cord. This fluid, which is normally water-clear, can be withdrawn by means of *lumbar puncture*, of which we shall have more to say later. Blood-stained fluid obtained on lumbar puncture of a patient who has just had a stroke always means that a cerebral hæmorrhage has occurred.

An absolutely instantaneous stroke may occur when a small piece of clot is swept into the circulation and sticks in one of the cerebral arteries (*cerebral embolism*). The clot usually comes from a diseased heart. Cerebral embolism is fairly common in mitral stenosis, when the clot comes from the left auricle; it may complicate mitral valvotomy. In coronary occlusion clots often form in the damaged heart, and here, too, bits may break off and cause cerebral embolism.

Treatment

The hemiplegic patient is a challenge to the nurses who are responsible for him; the frightful results of neglecting hemiplegia are to be seen in many an infirmary and home for the aged.

In the early stages the patient, if unconscious, is nursed on his side, in order to prevent inhalation of vomit. Turning him from one side to the other will help to prevent bedsores (p. 4) and hypostatic pneumonia.

If the patient is unconscious for more than a few hours he will need fluid, which may be given as a rectal glucose-saline infusion or as a subcutaneous infusion with hyaluronidase. (Hyaluronidase speeds up the absorption of fluid from tissues.) If there is retention of urine he must be catheterised every eight hours. Whenever repeated catheterisation is necessary the most rigidly aseptic technique must be observed, or cystitis is likely to occur.

As soon as possible the patient must be fed. Milk and other nourishing liquids can be given through a nasal tube as long as the patient cannot swallow. If he is constipated an enema should be given after a couple of days; purges are to be avoided.

Those who nurse hemiplegics must beware of three dangers—bedsores, hypostatic pneumonia and contractures. The prevention of bedsores and pneumonia, to which these patients are specially prone, is described in Chapter I. *Contractures* occur when the paralysed limbs are allowed to stiffen until they are completely fixed and rigid. Adhesions then form in and around the joints, and the patient is left with a painful, completely immovable limb. Such a disaster will never occur in a well-conducted ward. In such a ward the nurse in charge will put each paralysed limb through its whole range of

movement two or three times a day, starting on admission. As soon as the patient can move the limbs himself to the smallest extent he must be vigorously encouraged to do so. The good arm is put in a sling for part of each day so that the patient has to use the weak one. To exercise the fingers—as a rule the last part of the paralysed side to recover—nothing is better than modelling in plasticine. The patient is given a lump of plasticine and asked to model, say, a duck, with his weak hand. Warmly encouraged by the other patients in the ward he tries hard to produce a recognisable duck, and finds his weak hand is not as useless as he supposed. Those who cannot use plasticine may squeeze a hollow rubber ball in and out. As soon as possible he is got out of bed and encouraged to walk, at first with assistance, later alone or with a stick. He can often be helped by a walking iron with a toe spring, which corrects foot drop.

The *aphasic* patient is naturally most alarmed at finding himself dumb. Remember that he can understand all you say; reassure him whenever you can. Unlimited patience and understanding are necessary in face of his frantic and unavailing attempts to make his meaning clear. He may have to be taught to speak just as if he were a child, and a speech therapist is of the greatest help in this difficult and time-consuming re-education. Unfortunately, the aphasia is sometimes permanent.

Case Histories.—A woman of 33, while under treatment for heart failure due to auricular fibrillation and mitral stenosis, suddenly found she was unable to speak or to use her right hand; she did not lose consciousness. She was reassured and kept at rest; luckily the aphasia, which must have been caused by a small cerebral embolism, only lasted a few hours. Some weakness of the right hand, however, persisted.

Mrs. E., aged 55, had had a high blood pressure (220/115) for years, when she had a stroke. She was unconscious for two days and recovered consciousness to find herself paralysed down the right side and unable to speak. In spite of good nursing and other treatment she never was able to speak or move her right arm or leg again. Although bedridden and incontinent she was still, four years after her stroke, a most cheerful person, and had taught herself to knit with her left hand alone. She could only say "Yes," which she repeated over and over again, using different expressions and gestures to convey her meaning. She could read and understood everything that was said to her. When she was struggling to make known some request and someone at last understood what she meant and supplied the missing word she nodded and smiled, but could not repeat the word.

The Outlook in Hemiplegia.—This depends on the extent of the damage to the brain and on the standard of nursing. A cerebral hæmorrhage or a very widespread cerebral thrombosis is likely to be fatal within a day or two. Among those who recover consciousness

there will always be a few who are permanently disabled however good the nursing and the rehabilitation, but the great majority can be nursed back to a reasonably active life.

A person who has once had a stroke is always liable to have another, for the rest of his cerebral arteries are likely to be diseased and may at any time burst or become blocked by clot. Hemiplegic patients, therefore, should lead a quiet, regular life, avoiding sudden strain and heavy exertion, both mental and physical. Many of them can in this way live quite an active life for many years until the almost inevitable cerebral hæmorrhage finally carries them off.

A Note on the Nursing of the Unconscious Patient

Those nursing an unconscious patient, whether he is suffering from a stroke, meningitis, head injury, diabetic coma or poisoning, need exceptional vigilance and skill. Every normal bodily function—breathing, feeding, passing urine and stools—presents a problem. Because the patient cannot cough or swallow saliva he may inhale secretions, causing pulmonary collapse or hypostatic pneumonia; because he cannot move he is liable to develop bedsores and contractures. Since he cannot control his urine he is either incontinent (which increases the risk of bedsores) or suffers from retention, with the risk of urinary infection and renal failure. All these dangers are present in addition to those of the illness causing the unconsciousness; all require continuous attention and special treatment. The unconscious patient must never be left alone and must be under constant intelligent observation.

Breathing.—The unconscious patient will die if his airway becomes blocked by saliva, nasal or bronchial secretions or vomit. To prevent this he is nursed on his side, his position maintained by carefully arranged pillows. Often the foot of the bed is raised, which helps further to drain the air passages. The patient should be turned every two hours; this helps to prevent accumulations of secretions at the back of the throat, and also helps to prevent bedsores.

As well as attending to the patient's posture in these ways it is often necessary to aspirate secretions from the back of the throat. *An efficient suction apparatus should always stand by the bedside of the unconscious patient.* If he vomits the vomit must be aspirated immediately.

In deep coma it may be impossible to keep the patient's airway clear without passing an *endotracheal tube*. The doctor will do this via the nose or the mouth, using a laryngoscope. The nurse must keep the tube clear. The tube makes efficient aspiration of secretions

from the air passages possible. If the patient is likely to be deeply unconscious for more than 24 hours a tracheotomy is often performed; aspiration can then be carried out through the tracheotomy tube.

Feeding.—Unless he is likely to vomit, *tube feeding* is satisfactory, and the patient can be given adequate amounts of fluid and food, including milk, glucose, vitamins, etc. Tube feeding, however, is impossible if the patient is vomiting. Fluids must then be given either subcutaneously (plus hyaluronidase) or intravenously. Glucose saline can be given by either route; plasma (which provides protein) can only be given intravenously. *Rectal drips* are unsatisfactory, as little but fluid is absorbed, and much of the fluid is returned.

Bedsore.—The patient, as we have seen, must be turned every two hours. Pressure points are treated at the same time. Water-repellent barrier creams are helpful. Sheets must be changed frequently if the patient is incontinent.

Contractures.—Frequent changes of position also help to prevent contractures. Passive movements of the limbs are necessary as well.

Urine.—If retention occurs, eight-hourly catheterisation will be necessary, using the most scrupulous aseptic technique. (An indwelling catheter is sometimes preferred.) Urinary antibiotics are often prescribed to prevent the infection to which these patients are so susceptible. Sulphonamides, chloromycetin, tetracycline, and mandelates are all used; no one drug is given for more than a week for fear of inducing drug resistance.

Temperature and Pulse.—Record the temperature and pulse at least every two hours. In certain conditions, such as head injury, stroke and meningitis, hyperpyrexia may develop. In such conditions, therefore, it is often necessary to take the temperature every hour or half-hour. Tepid sponging is required if the temperature reaches 104° . A *falling pulse rate* is an important sign of rising intracranial pressure—e.g. in head injury. The *blood pressure* usually *rises* in such cases.

Other Observations.—The nurse must observe carefully, record accurately, and report promptly to the doctor any change in her patient's condition. Thus, cyanosis or respiratory difficulties; fits or twitching; changes in the level of consciousness and, in certain cases changes in pupil size, must all be reported, as well as alterations in temperature, pulse and blood pressure.

(2) Subarachnoid Hæmorrhage

In subarachnoid hæmorrhage the bleeding occurs, not into the

brain itself, but between the brain and one of its coverings or meninges—the *arachnoid membrane*. The cause of such bleeding is generally an inborn weakness of one of the arteries at the base of the brain. The weakened artery dilates and finally leaks blood or bursts.

The space between the brain and the arachnoid is normally filled with cerebrospinal fluid; hence, bleeding into this space results in bloodstained fluid as may be seen on lumbar puncture. The blood irritates the meninges, so that many of the symptoms of subarachnoid hæmorrhage are like those of meningitis (see p. 323).

Symptoms.—The victim of a subarachnoid hæmorrhage is usually a *young adult* who is suddenly seized by a blinding headache. He may rapidly become unconscious, when he will look much like the patient who has had a stroke except for the age difference and the absence of signs of hemiplegia. Such a patient may die within a day or two, or he may gradually recover consciousness. If he recovers he suffers from severe headache and stiffness of the neck; later on he may recover completely or be left with various kinds of paralysis.

In less severe cases the patient does not lose consciousness. Headache is severe and lasts a long time, and his neck is stiff and causes pain on bending the head forwards. The temperature rises a degree or two, and low fever lasts for a week or more; the pulse is usually slow. The urine often contains sugar and albumin.

Investigations. *Lumbar puncture* reveals cerebro-spinal fluid which is under increased pressure and often looks like pure blood. This generally enables the diagnosis to be made at the bedside. *Cerebral angiography* will be necessary if surgery is contemplated, for these special X-rays of the cerebral arteries will probably reveal the source of the bleeding. A substance such as diodone which shows up in an X-ray is injected rapidly into the *carotid artery* while a rapid series of X-rays of the head are taken.

Angiography is not without danger and can only be undertaken in a well equipped centre.

Treatment.—These patients need several weeks' absolute rest; morphia or pethidine should be given for the headache. All sudden, jerky movements must be avoided for fear of starting off the bleeding again. If the pulse rate is falling—a sign that the pressure inside the skull is rising—lumbar puncture may help to relieve the symptoms, but if too much fluid is withdrawn there is a risk of encouraging the bleeding.

Surgical treatment is sometimes advised; the results in expert hands are encouraging.

The Outlook in Subarachnoid Hæmorrhage.—A patient who recovers after a subarachnoid hæmorrhage is always in danger of having another. The risk of fresh bleeding is greatest during the first fortnight of the illness and, if this danger period is survived, the patient may live for many years without a recurrence.

The Cerebrospinal Fluid

Subarachnoid hæmorrhage is one of the many diseases in which a lumbar puncture is necessary for diagnosis. Here is a brief account of the *cerebrospinal fluid*, which is drawn off on such occasions.

Loosely covering the brain and spinal cord is a sheet of cobwebby tissue called the *arachnoid*—one of the *meninges* or membranes enclosing the central nervous system. The space between brain and cord and this membrane is filled with cerebrospinal fluid. Food substances pass from blood to brain via this fluid. The cerebrospinal fluid also protects the brain from injury, by acting as a cushion between it and the skull.

Examination of the cerebrospinal fluid tells us a great deal about the state of the nervous system. We have already seen that it is *bloodstained* in cases of subarachnoid hæmorrhage and, sometimes, after a stroke. In pyogenic meningitis (p. 323) the fluid is turbid or *purulent*, showing that the meninges are acutely inflamed. In other diseases (e.g. neurosyphilis, p. 295; tuberculous meningitis, p. 326) the fluid looks clear, but on examination is found to contain *too many cells* and *too much protein*—both signs of acute inflammation. (Compare the *pus cells* and *albumin* found in the urine when the bladder is inflamed, p. 186.) Other chemical tests may also reveal abnormalities which help in the diagnosis of nervous disease—e.g. the fluid contains *too little sugar* in most types of meningitis, and *too little salt* in tuberculous meningitis. In neurosyphilis the Wassermann reaction is generally positive.

(3) Poliomyelitis (Infantile Paralysis)

Poliomyelitis is an infectious disease, affecting chiefly children and young adults. It is commoner among those under 15, but more severe and lethal in adults. Since 1947, when 8,592 cases were notified, there have been several severe epidemics in this country. Epidemics usually occur in the late summer or autumn. Since the introduction of poliomyelitis vaccine a few years ago the incidence of the disease has been lower.

Symptoms.—The disease often begins insidiously, with a sore throat, nasal catarrh and slight fever lasting a day or two. This is

called the *prodromal stage* (prodromal = "running before"). Often this stage is absent and the first symptoms are those of the *pre-paralytic stage*. The patient suddenly feels ill, with pains in the spine, trunk or limbs; his head aches, his neck is stiff and he may feel sick or giddy; as a rule the temperature is raised. This stage lasts for several days, during which the patient gets steadily worse. He may then make a complete recovery, or the disease may go on to the *paralytic stage*. The paralysis comes on rapidly; it may be of any degree of severity, affecting muscles all over the body, or only one or two muscles of one limb.

The paralysis is at its worst within one to three days of its first appearance. The affected limbs are painful and both skin and muscles are acutely tender; muscle spasm is often severe at this stage.

If the respiratory muscles (intercostals, diaphragm and abdominal muscles) are affected the patient's respiration will deteriorate, and his life will be in danger. Such a patient is restless, cyanosed and distressed, with rapid, shallow respirations.

In another grave form of the disease the muscles of *palate and throat* are paralysed so that the patient cannot swallow. He is in danger of drowning in his own secretions should the unswallowed saliva and nasal secretions collect in the pharynx and trickle down into the trachea. (This type of the disease is called *bulbar paralysis*, because the nerves to the muscles concerned with swallowing come from a part of the brain sometimes called the *bulb*.) The most dangerous cases of all are those with both respiratory and bulbar paralysis.

In favourable cases of poliomyelitis paralysed muscles show some sign of recovery within a week or two. Muscle spasm and tenderness clear up, and the paralysed muscles are now seen to be flaccid. Reflexes, such as the knee jerk, are diminished or absent. Recovering muscles go on improving for several months, and the patient is left with much less extensive paralysis than he had in the acute stage.

The Cause of Poliomyelitis.—Poliomyelitis is caused by a virus which attacks certain parts of the nervous system. The virus can be found in the patient's throat during the first few days of his illness; it is present in the stools for several weeks or longer, and this is probably the main source of the infection.

The virus, once absorbed, finally reaches the spinal cord, where it singles out for attack those cells from which the *motor nerves* arise—those nerves which are responsible for the movement of muscles. A great many of the cells so attacked recover in the end—hence the recovery of muscles which are paralysed in the acute stage of the disease. If the motor nerves die the muscles they control waste and

Cause of the symptoms.—Shingles is caused by an infection of a little group of *nerve cells*, close to the spinal cord, from which arise the nerves to the affected area of skin. These cells constitute the



Fig 84 Shingles (Herpes Zoster) (Dr Richard Asher's case)

dorsal root ganglion. The infecting organism is either the virus of *chickenpox* or one closely related, for children may develop ~~chickenpox~~ ~~chickenpox~~

two weeks after contact with a case of shingles. It is rare, however, to catch shingles from chickenpox cases, and one disease does not protect against the other. (See Fig. 84, p. 305.)

Treatment.—Aspirin and codeine are given for the pain. The rash should be powdered and kept dry and covered to prevent secondary infection. Vitamin B₁₂ and tetracyclines have been recommended. Deep X-rays or even cutting the affected nerve root may be required in severe post-herpetic neuralgia.

(12) Polyneuritis

Neuritis means inflammation of a nerve. The term is much misused, and thousands of people attribute their aches and pains to "neuritis" when there is no reason to suppose that their nerves are inflamed.

Inflammation of many nerves, or *polyneuritis*, occurs in the most strangely diverse conditions—diphtheria, alcoholism, arsenic poisoning, diabetes, vitamin B₁ deficiency (beri-beri), pernicious anæmia, lupus erythematosus, cancer (especially lung cancer) and also in an acute illness called *acute febrile polyneuritis*. (This last may be caused by a virus.) In all these conditions some poison seems to injure widely scattered nerves, especially those of the limbs. The patient first notices tingling and numbness or weakness of the hands and feet. Later these symptoms spread up the limbs and, finally, the hands and feet may be completely paralysed and without sensation, and wasting of the muscles occurs. In very severe cases the muscles of respiration may be paralysed. In polyneuritis both motor and sensory nerves are affected, though one set of symptoms may be much more obvious than the other.

A curious feature of polyneuritis is that it is sometimes accompanied by signs of damage to the heart muscle, such as a rapid, feeble pulse. It seems that the heart is often susceptible to the same group of poisons as the nerves.

Treatment.—Cases of polyneuritis need prolonged bed rest and careful nursing, not only so that the paralysed muscles may recover, but because of the danger of heart failure. When the legs are affected *foot drop* is very apt to occur, and must at all costs be prevented by keeping the feet propped up with sandbags so that they are at right-angles to the legs and by supporting the bedclothes on a cradle. The weakened limbs must from the very beginning be put through their full range of movements daily to prevent contractures; later on massage and exercises will be required.

Apart from general nursing care, and the relief of pain by means of

aspirin and similar drugs, the only treatment that is required is to remove the cause of the neuritis, where this is known. The drunkard must be separated from his bottle, the beri-beri case plied with vitamin B₁ preparations, the diabetic has his insulin adjusted and so on. As vitamin B₁ is very successful in curing the polyneuritis in beri-beri it has been used in other kinds of neuritis with equal enthusiasm but little success, except in alcoholic cases, where the neuritis is probably due to vitamin deficiency, the result of the drunkard's poor appetite and digestion. Alcoholic neuritis responds well to large doses of vitamin B₁ by injection and by mouth; vitamin B₁₂ is often given as well.

(13) Disorders of Individual Nerves

In polyneuritis *many nerves* are injured. Here are some disorders of *individual nerves*.

(a) Trigeminal Neuralgia (tic douloureux)

The trigeminal nerve is the sensory nerve to the face. In trigeminal neuralgia, the patient, usually an elderly person, suffers lightning attacks of agonising pain on one side of the face, generally about the cheek and jaw. It is as if the face were suddenly pierced with red-hot wires. The pain may be brought on by cold winds, or by movements, such as chewing; often it occurs spontaneously. We do not know what causes this distressing malady, which has been known to drive the sufferer to suicide.

Treatment.—In mild cases sedative and pain-relieving drugs are prescribed. Vasodilator drugs may help; triline inhalations sometimes give relief. In severe cases the trigeminal nerve is destroyed by injecting alcohol into the ganglion just inside the skull from which it arises (the *Gasserian* ganglion); or the nerve may be cut at operation; unfortunately this causes permanent numbness of the face.

(b) Bell's Palsy

Here there is *paralysis of one side of the face* of sudden onset. Bell's palsy is a disorder of the *facial nerve*—the motor nerve to the face—but we do not know its cause. (The affected side of the face is motionless and expressionless; the corner of the mouth droops and the eye cannot be completely closed. Usually the paralysis clears up completely after a few weeks but in some cases it is permanent. No *curative treatment* is known. The patient should keep warm; salicylates are often prescribed. Physiotherapy is generally recommended after the first week or two, but is of doubtful value.)



Fig. 85 Bell's Palsy.
The left side of the face is paralysed (Dr Richard Asher's case)

(c) Sciatica

Sciatica is a symptom, not a disease. The name is applied to any condition where there is pain along the course of the sciatic nerve—the buttock, the back of the thigh, the back and outer side of the leg and the outer border of the foot. "Sciatica" is no more a diagnosis than "abdominal pain" would be; "What causes the pain?" is our first question in either case.

Anything which irritates or presses on the sciatic nerve may cause sciatica—arthritis or tumours of the lumbar spine, a misplaced intramuscular injection (p. 416). One of the most frequent causes of sciatic pain is now believed to be a *ruptured intervertebral disc*. This is what happens in such cases. Throughout the spinal column the vertebræ are separated by special discs, each of which has a hard

rim of cartilage and a soft, pulpy core. Sometimes, after an injury to the spine, this core bursts through the rim into the canal which encloses the spinal cord and nerve roots. A nerve root may be so stretched over the protrusion that it is seriously damaged and when, as is usually the case, the root involved is one of those from which the sciatic nerve arises, the result is sciatica. A *prolapsed disc* may be the result of an injury; the patient may, for instance, have strained his back while trying to lift a heavy weight. The strain is followed by back-ache—often very severe—and the pain gradually extends down one leg.

The pain is made worse by bending the spine, by lifting heavy weights and often by coughing, sneezing or straining at stool. In severe cases foot movements may be weak, and there may be some loss of sensation over the foot and outer side of the leg.

Treatment.—Mild cases of sciatica clear up after a week or two in bed. In more severe cases several weeks in bed are necessary; boards should be placed under the mattress to prevent sagging. Traction on the affected leg may help. When the patient gets up he should wear a surgical belt designed to support the lower spine and prevent excessive movement of the spine, or a plaster jacket may be worn. Sometimes the pain responds to this restriction of spinal movements without any period of bed rest. In very severe cases, where the pain does not respond to these measures, the prolapsed disc may be removed at operation (laminectomy). Operation is, however, advised less frequently at present than formerly.

Massage and exercises will be necessary during convalescence, whether medical or surgical treatment has been adopted. If sciatica is secondary to some other disease the latter must, of course, be treated,



Fig. 86. The Sciatic Nerve. This nerve runs down the back of the thigh and leg, it supplies part of the skin of the foot and leg (shaded in diagram) and the muscles of the back of the thigh, calf and those which move the foot and toes.

(14) Ménière's Syndrome

Sudden attacks of giddiness in a middle aged person who is going

deaf in one ear are characteristic of Ménière's syndrome. The attacks occur at irregular intervals, and are often very severe; the patient feels that the room is turning round—the sensation is quite different from "dizziness" or faintness. During the attacks nausea and vomiting are common.

The deafness usually starts in one ear, though both may finally be affected. Buzzing and ringing noises in the head (tinnitus) are common, and may be troublesome before the deafness is noticeable.

The *cause* of Ménière's syndrome is disease of the inner ear, which contains both the organ of *hearing* and a complicated structure—the labyrinth—concerned with the sense of *balance*. In Ménière's syndrome the labyrinth may be dilated with excess fluid.

Treatment.—Symptoms may be relieved by sedatives, such as phenobarbitone, or by anti-histamines. Salt restriction may help. Often the attacks of giddiness clear up after a time, but if they are persistent and crippling an operation in which the labyrinth is destroyed may be necessary. This may destroy the remaining hearing in that ear, but this is often preferable to continued, disabling attacks of giddiness.

(15) Epilepsy

✓ Epilepsy is a condition in which the patient is subject to fits—attacks which come on for no known reason; these attacks most often consist of impaired or altered consciousness, or muscle twitching, or both.

We have already come across several diseases in which fits may occur—high blood pressure, uræmia, insulin overdosage, brain tumours and neurosyphilis, to mention a few. Fits may also occur after a head injury, in any high fever in infancy and in many other conditions. In the majority of cases there is no obvious cause for the fits; the condition is then known as *idiopathic epilepsy*.

Epilepsy tends to run in families; among the near relations of epileptics there are three times as many epileptics as among the relations of normal subjects. Migraine and epilepsy are often found in the same family.

Epilepsy is a very common condition, affecting perhaps one in 200 of the population.

An epileptic history.—Epileptic attacks generally start in childhood or adolescence; if the first fit occurs after the age of 25 some cause such as brain tumour or abscess is suspected—though often no such cause is found. In most cases it is impossible to say what caused the first fit, which may occur when the child seems to be in good

health and spirits. Of course, if any illness, mental shock or fall on the head has recently occurred the parents are sure to blame it for the occurrence of fits.

For the rest of his life the patient, if untreated, is liable to have fits at any time. Some epileptics have attacks every day, others go for months without a fit and then have a very severe one. Some patients always have their fits by day, others by night; others have several fits in one day at more or less regular intervals of one to four weeks.

Epileptic Attacks.—The two commonest varieties of epileptic attack are the *major fit* (*grand mal*) and *minor epilepsy* (*petit mal*). Most epileptics have both kinds of attack at different times.

Attacks may come on at any time, but they are rare when the patient is at work or interested in what he is doing. An epileptic may know when an attack is coming on by unusual feelings of restlessness, headache, dreaminess, giddiness, sinking feelings and other sensations.

Major Fits.—The attack comes on suddenly, though twitching or spasm of a group of muscles may herald the general convulsion. The patient rapidly loses consciousness and falls to the ground, most often on his back. The back is arched, the head and the staring eyes are turned to one side, the teeth clenched and the limbs rigid—arms bent and held close to the side, the legs straight. As he falls he may give an unearthly cry, he may bite his tongue and he may pass urine. For a few seconds the whole body is fixed and rigid; no breath is drawn, so the patient goes blue in the face. After a few seconds the body begins to jerk violently, breathing is resumed and froth may ooze from nose and mouth. A minute or two later the patient lies limp, deeply unconscious and breathing quietly; as a rule he comes round a few minutes later, but for several hours he may be dazed and sleepy, and later he may be unable to remember what happened during this period.

Status epilepticus.—Here the patient has one convulsion after another without recovering consciousness; this may go on for hours or days. In the absence of effective treatment the patient may die of exhaustion or from the inhalation of vomit or saliva.

Minor Epilepsy (petit mal).—In these attacks the patient's consciousness is impaired for a few seconds. For a moment he looks blank and vacant, but he does not fall or drop whatever he is holding. After a moment he recovers, and may seem dazed, or he may go on as if nothing had happened. Some patients have dozens of such

attacks a day. In more severe cases the patient falls and loses consciousness, but convulsions do not occur.

Other forms of epilepsy.—Epileptic attacks may take many other forms: twitching or spasm of the face or one hand, peculiar feelings in the skin, an unpleasant taste in the mouth, dreamlike states, peculiar behaviour. Such attacks, with little or no disturbance of consciousness, may be the only symptoms of epilepsy, or the patient may also have convulsions or *petit mal*.

The Mental State in Epilepsy.—Most epileptics are mentally normal, especially if successfully treated. A few who have very numerous fits may become spiteful, mischievous and irritable.

Diagnosis of Epilepsy.—This is usually made on the history; a description of the fit by an intelligent observer is invaluable. Electroencephalography may help in doubtful cases, as the E.E.G. is often abnormal in epilepsy. Full neurological investigations are sometimes necessary, especially in older patients, to exclude organic disease.

Treatment.—Every effort must be made to fit the patient for a normal active life in the home, at school and at work. He need only avoid such activities as would be dangerous if a fit occurred, such as driving, sailing or mountaineering. Swimming should only be allowed if the epileptic is accompanied by someone who knows he is an epileptic.

Epileptic fits can generally be controlled by one or more of the following drugs: phenobarbitone (gr. $\frac{1}{2}$ –1), phenytoin sodium (“*epanutin*”) gr. $\frac{3}{4}$ –1 $\frac{1}{2}$, or primidone (“*mysoline*”) 250 mgm. One to three doses a day are necessary. If the patient always has his fits at some special time of day they may be controllable by a single daily dose—for instance, fits at night may be prevented by a grain of phenobarbitone in the evening. None of these drugs have much effect in *petit mal* which often responds best to troxidone (“*tridione*”) (gr. 1 $\frac{1}{2}$ –6). A careful watch for toxic effects must be kept, especially at the start of treatment. Phenobarbitone may cause depression and sleepiness, phenytoin, unsteadiness, troxidone and primidone, aplastic anæmia.

Treatment of the Epileptic Attack.—No special treatment is needed except to keep the patient from harming himself and to see that his breathing is not obstructed by tight clothes. A spoon is sometimes forced into the mouth in an attempt to prevent tongue-biting; this seldom does any good, because the tongue is bitten, if at all, the moment the jaws clench, at the very beginning of the attack.

Treatment of Status Epilepticus.—Repeated doses of paraldehyde

(5-10 ml.) or sodium phenobarbitone (gr. 3) are given by intramuscular injection. The nursing care of the patient is of vital importance (see "Nursing of the Unconscious Patient", p. 288).

The outlook for the epileptic.—The great majority can be treated so that they can enjoy a more or less normal life. In a few cases the fits are not controlled by treatment, and the patient goes downhill in mind and body.

(16) Migraine (Sick Headache)

The subject of migrainous attacks is often an unusually intelligent, able person who, from adolescence to middle age, suffers from repeated attacks of severe headache and sickness. Migraine often occurs in the same families as asthma, hay fever, urticaria and epilepsy. Attacks are usually more frequent when the patient is over-tired, or worried; in women attacks may be most frequent during the week or ten days before the menstrual period.

The Migrainous Attack.—This may be of any degree of severity from the insignificant to the disabling. Before the attack the patient may feel particularly well, or he may be unaware that an attack is pending. In a severe attack he may wake in the morning to find that, as soon as he lifts his head from the pillow, he is smitten with giddiness. For a short time he may lose half his field of vision (or see coloured lights or stars dancing before his eyes). Soon a throbbing headache begins and rapidly becomes intense; most often it is limited to one side of the head, but it may be generalised. Noise, movement and daylight all make the pain worse, and, in severe cases, all the patient can do is to lie motionless in a darkened room. He feels sick and often vomits repeatedly; he looks ill, with a drawn, greyish face. The attack lasts several hours, or even all day; once it is over the patient goes to sleep and wakes feeling, perhaps, shaken, but free of pain and sickness.

The cause of the symptoms is a contraction of certain intracranial arteries followed by dilatation. The contraction causes the visual disturbances; the dilatation, the throbbing headache. The cause of the arterial disorder is unknown.

Treatment.—*During the attack* the patient should be completely at rest and undisturbed in a darkened room. Pain relievers such as aspirin may be prescribed, but they are ineffective in severe cases. *Ergotamine tartrate* ("femergen") is most successful in some cases, and may cut short an attack. It is given in doses from 0.5 to 2 mgm., by mouth, injection, or suppository.

It is difficult to prevent attacks in migrainous subjects. They should live a healthy life, avoiding overwork and worry. Sometimes sedatives, such as phenobarbitone, taken regularly for weeks at a time, are helpful. When attacks are clearly associated with mental strain and anxiety, psychotherapy may be advised. As a rule, but not invariably, the attacks cease spontaneously during middle age—in women, about the time of the menopause.

A Note on Headache -

Headache is one of the commonest of symptoms. The pain may be trivial or devastating, and the cause may be a minor upset of the health or a mortal illness. Here are some of the more important causes of headache:

(1) *Abnormal tension* of the muscles of the scalp and neck. This muscle tension may be the result of mental tension and worry. This type of headache is common. It responds to mental and physical relaxation, massage, sedatives and pain relievers such as aspirin.

(2) *Migraine* is described above. The pain is due to dilation of intracranial blood vessels.

(3) *Fever*, as in influenza. The headache of fever is probably due to distension of intracranial arteries.

(4) *Drugs and poisons*, including the well-known alcoholic "hang-over". Here, too, distension of arteries probably occurs.

(5) *Hypertension*.—Headache often occurs in severe cases of hypertension. Headache in hypertension is sometimes due to worry and to the fact that "everyone" knows that "blood pressure" causes headache. Many hypertensives do not, in fact, have headaches if unaware that the blood pressure is high.

(6) *Brain tumour, cerebral abscess and meningitis* all cause raised intracranial pressure, and, hence, pain. Meningitis also causes pain because the inflammation involves pain-sensitive structures such as vessels and nerves. *The brain itself is insensitive to pain.*

(7) *Headache following Lumbar Puncture*.—This is associated with *lowered* intracranial pressure; the exact cause of the pain is unknown. If cerebrospinal fluid continues to leak from the puncture site the headache is likely to be more severe. For this reason the narrowest possible lumbar puncture needle is used. After lumbar puncture the patient should lie flat in bed, and should drink plenty of fluid. Extra salt is also helpful. If headache occurs, raise the foot of the bed.

An exceedingly severe headache may also follow ventriculography; morphia may be necessary to control it.

(8) *Head injury* may be followed by severe and prolonged headache.

(9) *Sinusitis*.—The nasal sinuses are cavities in certain of the bones of the face and base of the skull. They are lined with mucous membrane and communicate with the nasal passages. If this membrane is inflamed the opening into the nasal passage is liable to become blocked, and the sinus becomes distended with secretions and/or pus. This causes pain, which may be felt over the affected sinus (e.g. in the cheek bone, or just over the inner part of the orbit) or as a more generalised "headache".

(10) *Eye Disease*.—This has been placed last, though the public would probably put "eye strain" tied with "blood pressure" at the top of the list. Eye disease *can* cause headache, but in this writer's experience rarely does so. Eye strain usually causes discomfort in the *eyes*, especially after reading. The pain of diseases such as iritis and glaucoma is felt first and foremost in the eye though it may spread to the forehead and the rest of the head.

To sum up: a person who is "subject to headaches" usually has migraine, or muscle tension—the tense muscles reflecting a tense personality. The other conditions listed above are much less common as causes of *recurrent* headache. They may be suspected if a patient is attacked by headaches, never having had them before. Full investigation may be necessary, but the patient's description of his symptoms is always the first, often the most important and sometimes the only signpost to the diagnosis.

(17) Porphyria

This uncommon disease may be described here, although a disorder of the nervous system is only one of several forms.

Porphyria sometimes runs in a family. It may cause any of the following disorders: peripheral neuritis; mental confusion; a rash which is worse after exposure to sunlight; severe abdominal pain. Symptoms are sometimes brought on by drugs, especially barbiturates, to which these patients are very susceptible.

In porphyria abnormal substances called porphyrins are excreted in the urine. They sometimes make the urine *red in colour*, or the urine may *turn red on standing*. Sometimes the presence of porphyrins can only be demonstrated by special tests.

Treatment is symptomatic. It is important that the patient should know that barbiturates and other drugs may be dangerous.

CHAPTER TWENTY

INFECTIONS AND INFESTATIONS

WE have already come across several diseases which can be passed on from one person to another—e.g. infective hepatitis (p. 174), poliomyelitis (p. 291) and tuberculosis (p. 108). The illnesses described in this chapter are for the most part more infectious than these, and if hospital treatment is necessary they are often nursed in fever hospitals or special wards in general hospitals.

Many of them are *notifiable diseases*. This means that the doctor treating the case must *notify* it to the medical officer of health, who can then take any steps he may think necessary to stop the spread of infection. Thus he can arrange for the removal of the patient to a fever hospital, or for swabs to be taken from contacts, or for preventive inoculations to be given. Of the diseases so far described, pneumonia, poliomyelitis and tuberculosis are notifiable; nine notifiable diseases are included in this chapter—typhoid fever, dysentery, cerebrospinal meningitis, diphtheria, scarlet fever, erysipelas, whooping cough, measles and smallpox.

(1) Typhoid Fever

The almost complete eradication of typhoid fever from most civilised communities is one of the most enthralling chapters in the history of public health. From being one of the commonest of infectious diseases it has become so rare that an epidemic consisting of a few dozen cases is now headline news. This remarkable decline is due entirely to better sanitation; a man can only contract typhoid by taking food or drink contaminated, directly or indirectly, by the faeces of a typhoid case or carrier (p. 320). The worst epidemics are usually caused by defective drains, which allow sewage to seep into the water supply.

Symptoms.—The disease begins insidiously; the patient feels out of sorts, with a severe headache, cough and nosebleeds; either constipation or diarrhoea may occur. After a day or two he takes to his bed; each evening his temperature is a little higher, until, by the end of a week, it is 103° or more. (See p. 17.) The pulse rate is comparatively slow—perhaps below 100; the urine, as in most fevers, is scanty, highly coloured and contains albumin.

During the second week the temperature is maintained, the pulse

rate rises, and the patient's general condition deteriorates. Diarrhœa is often severe, with the passage of frequent, liquid, greenish-brown stools, though some patients are constipated throughout. The abdomen is distended and the patient wastes rapidly. His tongue and lips are dry, cracked and brownish. He often lies on his back, muttering and picking at the bedclothes in a quiet delirium. In severe cases the breathing is quickened and the cheeks and lips are bluish.

A characteristic symptom of typhoid during the second week is a rash consisting of *rose-coloured spots* which appear in crops here and there about the back, chest and abdomen.

In severe cases the patient's condition during the third week is still grave, and the most serious complications are apt to occur during the second and third weeks of typhoid fever. In most cases, however, the third week sees the patient on the way to recovery. The mists clear from his clouded brain, his appetite returns, the tongue becomes cleaner, diarrhœa abates and the temperature falls by degrees. The pulse rate may still be raised long after the temperature has fallen to normal.

Although complications and relapses may still occur, the patient usually gets better from now on. Naturally he feels extremely weak when he first gets up, but given a good diet he soon gains weight and strength. The pulse tends to be rapid, and swelling of the ankles may occur during convalescence.

The cause of the symptoms.—Typhoid fever is an infection with bacilli (*Salmonella typhi*) which, entering via the bowel, spread all over the body and circulate in the blood stream. The fever and prostration are due to the general infection. The abdominal distension and diarrhœa are due to the infection of the small intestine with typhoid bacilli. The lower reaches of the small intestine—the *ileum*—are the chief seat of attack, where the bacilli cause ulcers which may eventually penetrate the whole thickness of the intestinal wall.

Complications of Typhoid Fever.—As the typhoid bacilli circulate in the blood every organ may be attacked, and the complete list of recorded complications in this disease is an immensely long one, including pneumonia, cholecystitis, meningitis, nephritis, cystitis, bone abscess and femoral thrombosis. The commonest and most important complications, however, are *intestinal hæmorrhage* and *perforation*. Hæmorrhage into the bowel may occur from any of the many ulcers present. The patient suddenly feels faint, the temperature falls and the pulse rate rises; a little later he passes large quantities of bright blood mixed with fæces.

Perforation causes 20-30 per cent. of typhoid deaths. It may occur during the second or third week of the disease, an ulcer having penetrated the entire thickness of the bowel wall. Intestinal contents



Fig 87 Part of Bowel cut open to show Typhoid Ulcers

gush out into the peritoneal cavity. The patient has a sudden attack of shivering, abdominal pain and tenderness. The temperature and pulse rate both rise, and peritonitis occurs in the absence of prompt treatment.

Investigations.—The symptoms of typhoid vary from case to case, and often the diagnosis can only be made with the help of special investigations. During the first week the typhoid bacilli, which are circulating in the blood, may be found on *blood culture*. During the second week blood culture is generally negative, but the organisms can be found in the stools and, quite often, in the urine. The *agglutination reactions* are often helpful at this stage. The patient's blood will by now have developed a certain power of overcoming typhoid bacilli, and this is shown by the fact that his serum will now cause such bacilli to clump together or *agglutinate*. If the serum agglutinates typhoid bacilli even when it is highly diluted—to 1/100 or more—the case is almost certainly one of typhoid fever. (Widal reaction.) A white count shows a *leucopenia* (white cells fewer than normal), unlike what is found in most acute infections.

Treatment.—The patient's life is often in the hands of his nurses. Though chloramphenicol has been found to hasten recovery good nursing is still one of the most important parts of the treatment. (Those nursing typhoid patients should be inoculated against typhoid.)

The patient generally lies most of the time on his back; his position should be changed from time to time to prevent bedsores and hypostatic pneumonia, both of which are very apt to occur. He should be sponged twice daily, and his mouth must be gently cleansed after each meal—the most frightful infections may arise in the dry, dirty mouth of the neglected typhoid patient. The temperature, pulse and respiration rates must be charted at least four-hourly, otherwise complications, such as hæmorrhage, with its rising pulse rate, may be missed.

The typhoid patient is inclined to waste away, so he should receive as nourishing a diet as his inflamed bowel can digest. He is generally given some three pints of milk a day, in the form of milk drinks, custards and junket. Unless he is very ill he may also be given milk puddings, eggs and sieved vegetables. If there is constipation a plain saline enema may be given every second or third day; exhausting diarrhœa may be checked by means of opium preparations, such as tinct. opii. minimis 10.

Chemotherapy.—Chloramphenicol (chloromycetin) shortens the illness in the majority of cases. At first the dose is about 3 grammes daily. Often there is no apparent response for 48 hours, when there is a sudden dramatic improvement. The temperature falls, bowel symptoms clear up, the appetite returns and the patient becomes convalescent. Treatment continues with smaller doses of the drug

for several days at least; some advise a much longer course of treatment to guard against relapses which are otherwise common. Prolonged rest and careful observation of the patient are essential, during convalescence, for perforation and hæmorrhage may occur in an apparently convalescent patient.

Treatment of complications.—*Perforation* requires immediate operation.

Hæmorrhage requires absolute rest with the head low, the administration of morphia gr. $\frac{1}{4}$ and immediate blood transfusion if bleeding is severe.

Prognosis.—With modern treatment the mortality is about 5 per cent. Patients may die of general toxæmia and heart failure during the second or third week, or perforation, hæmorrhage or some other complication may carry them off. The 95 per cent. who survive generally make a complete recovery after a long illness. About 2–5 per cent. of these patients become carriers. One attack of typhoid usually gives lifelong immunity.

Typhoid carriers.—Carriers are people who carry disease-producing germs in their bodies, although they are not suffering from that disease. A person who has had typhoid may continue to pass infected fæces or urine for months or years. Unfortunately chloramphenicol does not rid carriers of typhoid bacilli. The patient should be informed of his condition, and warned against any occupation involving food handling which would make him a danger to others. He should beware of contaminating his hands, which are often the vehicles of infection, and should wash them carefully after using the W.C., drying them on his own towel. *Cholecystectomy* usually cures the carrier state, by removing the infected gall-bladder which is the main source of typhoid bacilli.

Prevention of Typhoid.—As we have seen, the best preventive against typhoid is an efficient water and drainage system. The spread of the infection in hospitals where typhoid patients are treated is prevented by scrupulous *barrier nursing* (p. 42). The patient's urine and fæces must be mixed with an excess of strong carbolic acid and allowed to stand for several hours before being thrown down the drain, for these excreta may be swarming with typhoid bacilli. Bed-pans and urine bottles must be sterilised, and everything used by the patient, such as spoons and plates, should be boiled.

Prophylactic inoculations.—Troops serving overseas, or anyone who is exposed to the risk of contracting typhoid fever may be protected by *inoculation*. We have seen that an attack of typhoid

fever generally leaves the person *immune* to further attacks. We can give him the same sort of immunity, though less complete, by giving him injections of *dead typhoid bacilli*. The body reacts to these bacilli just as if they were alive, and protective substances called *antibodies* appear in the blood stream which enable the person to resist the onslaughts of *living* bacilli, should he be exposed to typhoid infection. (These antibodies are similar to the *agglutinins* described above, which are produced during the second week of an attack of typhoid fever.) Typhoid inoculations are often referred to as "T.A.B.", as they usually consist of a mixture of the bacilli of typhoid and two similar but milder diseases called paratyphoid A and B. Two injections are usually given, 7 to 10 days apart. Further injections are necessary every two years as long as the person is exposed to infection.

The immunity which follows an attack of typhoid fever, or a series of typhoid inoculations, is called *active immunity*, because the antibodies are actively produced by the person's own defence mechanisms. An example of *passive immunity* is described on p. 336.

(2) Dysentery and Food Poisoning

A mild form of *dysentery* is extremely common in this country, and has become more so in recent years. It is as well for nurses to know something of this condition, because they themselves are liable to contract various diarrhoeal infections. A large proportion of the population are carriers of dysentery, and isolated cases or small outbreaks of the disease are common everywhere, especially in institutions.

The chief and sometimes the only symptom of dysentery is *diarrhœa*. In severe cases the temperature rises and numerous fluid stools are passed, perhaps containing blood and mucus. In such cases, the patient may be very ill, especially if he is old, or very young. Vomiting is rare, except at the onset. Mild attacks, in which 4 or 5 loose stools are passed daily for a few days, are very common, and often seem too trivial to send the patient off duty.

Dysentery is caused by an infection of the colon; in this country the *Sonne bacillus* is usually responsible. The organisms are present in the stools, and the disease is spread in much the same way as typhoid. In the interest of the patients, therefore, any nurse who has diarrhœa should report sick. Of course, there are many causes of diarrhœa besides dysentery, but the latter should always be suspected if several cases occur in a community.

The spread of dysentery may be to some extent prevented if the

hands are always washed after using the W.C., and before cooking, serving, selling or eating food.

Patients with moderately severe dysentery are best treated in bed. A light, nourishing diet is allowed with plenty of fluid, and a course of sulphaguanidine or succinyl sulphathiazole prescribed—9–12 gm. a day for several days. These drugs are sulphonamides which remain in the bowel, instead of being absorbed; they cure many cases of dysentery by killing the bacteria in the gut. Unfortunately, many strains of dysentery bacilli are now sulphonamide resistant. In these cases oral streptomycin, tetracycline or chloramphenicol may be used. If the patient is ill and severely dehydrated, intravenous fluids will probably be necessary.

Food poisoning.—This is a much more acute disease; it is caused by the eating of food contaminated with food poisoning organisms or their toxins. (Most of these organisms are members of the *Salmonella* group, like the typhoid bacillus. Staphylococci and many other organisms can, however, cause food poisoning.) As a rule, everyone who has partaken of some particular dish is afflicted more or less simultaneously with severe vomiting, diarrhoea or both, within a few hours of the meal. Although the victims may feel extremely ill for a day or so, severe infections are very rare, and no special treatment is required apart from seeing that the patient is warm and comfortable and has plenty of fluid to drink.

An outbreak of food poisoning must be reported to the Medical Officer of Health, who then takes steps to find the source of infection.

History of an outbreak of dysentery.—One Monday morning in February several general practitioners were surprised to find that most of their calls were to patients with severe diarrhoea. They rang up their colleagues in the district and found that their experience was the same. They immediately rang up the Medical Officer of Health, who sent his officers to visit afflicted households within 24 hours and to take specimens of faeces. These officers asked each patient about what food he had bought recently, about meals out, and about his milk, bread, grocery and meat suppliers. The majority of patients patronised the same grocer. This grocer was then visited and asked if any of the assistants had diarrhoea. As far as he knew they had not. The assistants were then asked if they would mind specimens of faeces being taken for culture. Somewhat reluctantly they agreed. One girl was found to be a *Sonne* carrier—the same strain as was found in the faeces of dysentery cases. This girl was sent off duty and treated with succinyl sulphathiazole; she was not allowed back until her stools were negative. (She had never had any diarrhoea.)

This outbreak lasted for several weeks; often whole households were laid low. Fortunately very few people were seriously ill. Cases occurred here and there throughout the spring and early summer, when a second

outbreak occurred, centred around a few streets about a mile away. The source of the second outbreak was not identified; probably it was a case or carrier resulting from the first outbreak.

(3) Meningococcal Meningitis (Spotted Fever)

The history of this remarkable disease during the past forty years is an astonishing one. Cerebrospinal meningitis is quite a modern disease. Almost unknown during the nineteenth century, the first important epidemic occurred in Glasgow in 1907-8. It has been fairly common ever since, but the incidence shot up during both world wars, and thousands of civilian and Service cases occurred in 1915-17 and 1939-41. No less dramatic than this rise in incidence, at a time when practically all other infectious diseases are becoming less common, has been the taming of this highly fatal disease by the sulphonamides. Originally 50-70 per cent., the general mortality is now about 15 per cent., and with really first-class medical and nursing treatment the death rate can be kept below one case in twenty.

Meningococcal meningitis is not very infectious, and it is quite safe to nurse cases in a general ward, the usual barrier nursing precautions being observed. The disease is spread chiefly by carriers in whom the responsible organisms flourish in nose and throat. These carriers, unlike those of typhoid fever, are not usually people who have had the disease.

The infection is spread by the inhalation of infected droplets; hence overcrowding and lack of ventilation favours outbreaks of meningitis. It has been found that when more than 20 per cent. of a community have become carriers an outbreak of meningitis is likely.

Symptoms.—The onset of meningitis is generally acute, the patient, often a child or young adult, being suddenly smitten with headache, fever and vomiting. The headache is very severe and is felt chiefly in the back of the head. In infancy the illness may start with convulsions.

For the first day or two there may be little to distinguish such a patient from one suffering from influenza or some other acute fever. As the disease develops its true nature becomes apparent. The patient lies curled up on his side, huddling under the bedclothes to avoid the light. He is restless and irritable and resents disturbance; in severe cases he may be delirious, but even in his delirium he is tormented by his aching head, and vomiting continues. (In most fevers the delirious patient loses his headache.) The temperature may be high— 103° or more—the pulse slow and often irregular. The neck is stiff and painful, and the patient cannot bend his head

forward without pain. The muscles bending the drawn-up knees are in spasm, and the leg cannot be lifted with the knee straight—an important sign of meningitis called *Kernig's sign*.

During the first week various rashes may appear. The most characteristic consists of those purple spots from which the disease derives its name of spotted fever. Herpes of the lips may occur, as in other infections.

Sometimes the disease adopts a far more malignant form than that described above. Rapid prostration may occur without any special signs, and the patient may die within 24 hours.

In other cases—especially in infants—the disease begins insidiously, so that the diagnosis is not made for several days. In general, the younger the child the less distinctive the symptoms.

Investigations in Meningitis. The most important of these is *lumbar puncture*, which in most cases enables the diagnosis to be made at the bedside. The cerebrospinal fluid is under increased pressure and may gush out of the lumbar puncture needle. The fluid looks cloudy and yellowish or even frankly purulent; microscopic examination shows that the cloudiness is due to the presence of millions of pus cells. The organisms causing the meningitis—meningococci—can usually be demonstrated. Meningococci may also be cultured from the blood.

The Cause of the Symptoms.—In cerebrospinal meningitis the membranes covering the brain—the *meninges*—are infected with germs called meningococci. These inflamed membranes pour out masses of fluid which raises the pressure inside the skull; we have already seen (p. 300) that raised intra-cranial pressure may cause headache, vomiting and slow pulse. The pus cells make the cerebrospinal fluid turbulent, as seen on lumbar puncture.

If the infection is not quickly overcome by treatment the inflamed membranes may stick together round the base of the brain, so the fluid is dammed back; this causes the *post-meningitic hydrocephalus* which may occur in infants.

The *rash* in meningitis is caused by meningococci circulating in the blood. When rapid and severe prostration occurs, this usually means that the *adrenal glands* have been affected; post mortem of these glands often show hæmorrhages. The symptoms are those of acute adrenal insufficiency (p. 235).

Treatment.—The patient with meningitis must be nursed with the care and skill that are always necessary in cases of severe fever. Feeding may be a difficulty in a severe case, and if the patient cannot swallow he will have to be fed through a nasal tube. (See

p. 288 for the nursing of the unconscious patient.) Constipation and retention of urine may occur, necessitating enemata and catheterisation.

The infection of the meninges can, in the majority of cases, be cured by large doses of sulphonamide. One of the more powerful drugs is chosen—sulphadiazine or sulphadimidine—and enormous doses are often necessary to start with—12 gm. a day or more for an adult. As usual, such treatment necessitates a large fluid intake, which must be given intravenously if the patient cannot swallow. The urine must be kept alkaline—e.g. by giving citrates.

Penicillin is an even more powerful meningococcus-killer but, unfortunately, it does not filter through into the C.S.F. as do the sulphonamides, and if it is to be effective in meningitis it must be given by lumbar puncture or even into the ventricles of the brain through a burr-hole in the skull.

A single intrathecal injection of penicillin is often given at the first lumbar puncture, when purulent fluid is found but before the organism has been identified. Repeated intrathecal injections of penicillin are necessary in certain types of pyogenic (pus forming) meningitis, but if meningococci are found further penicillin injections will be unnecessary except in very severe cases.

In fulminating cases with circulatory collapse steroids are necessary, and intravenous solutions containing salt and glucose are given.

Treatment of Symptoms.—Headache may be so severe that morphia or pethidine is needed to control it. Repeated lumbar puncture is occasionally helpful, as it relieves the raised intracranial pressure by letting off some of the excess fluid. Phenobarbitone is given to control restlessness or prevent fits.

Results of Treatment.—Unless treatment is delayed or inadequate or the infection is exceptionally severe, meningococcal meningitis can nearly always be rapidly cured, and the patient may be out of hospital in less than three weeks. Nothing is more dramatic than the recovery of such a patient, who may have been moribund on admission. Many avoidable deaths occur owing to misdiagnosis and delay in starting treatment, especially among infants.

Complications.—The most important is deafness, which is caused by damage to the auditory nerves, and unhappily is apt to be complete and permanent. More cases of post-meningitic deafness are seen to-day than in the past, for these patients would not have lived to become deaf in pre-sulphonamide days. Young infants with meningitis, especially if treatment is delayed, may become mentally defective. Spastic paralysis (p. 296) may also occur.

Meningococcal septicæmia.—It was pointed out above that the meningococci cause a generalised infection of which meningitis is but a part. This is shown by the development in certain cases of relapsing fever, acute arthritis and a curious rash consisting of raised pinkish spots and lumps. Meningitis may or may not occur in such cases, but the meningococci can generally be obtained on blood culture. Intramuscular injections of penicillin will clear up those infections in which the meninges are not involved.



Fig 88 Meningococcal Septicæmia
Rash accompanied by irregular fever in a man of 29. Meningococci on blood culture. Cured by penicillin.

Other Forms of Meningitis

The meningococcus is by no means the only organism which may attack the meninges, though other forms of meningitis are less common.

Tuberculous meningitis is a much less acute disease; it most often attacks infants and children who are exposed to heavy infection by being in contact with an open case of consumption. The early symptoms are vague; the child seems unwell and is apathetic yet

irritable. He may have a headache, and is often sick and constipated. After a week or two the child sinks into a deep stupor and lies motionless, gazing into infinity with wide vacant eyes. The C.S.F. is clear or opalescent, but it is under increased pressure and contains an excess of cells and protein. The diagnosis is made absolutely certain if tubercle bacilli can be found in the fluid. Until streptomycin was discovered, tuberculous meningitis was invariably fatal; a large proportion of cases may now be cured if the diagnosis is made early and treatment started at once with anti-tuberculous drugs.

A combination of streptomycin and isoniazid has proved very effective in the majority of cases. Daily intramuscular injections of streptomycin are given. Some physicians still advise that streptomycin should also be given intrathecally, at least at the beginning of treatment, but this is less common now. Chemotherapy must be continued for at least a year. *Cortisone* is sometimes given, especially if a block in the circulation of C.S.F. is suspected; such a block is due to adhesions in the inflamed meninges. Injections of tuberculin (P.P.D.) are occasionally given for the same reason. The outlook is good except in infants and very young children, among whom most of the deaths now occur. Infants who recover from tuberculous meningitis may be mentally defective.

(b) Meningitis caused by Pus-forming Organisms

Here the meninges are infected via the middle ear, or other local septic focus; sometimes the meningitis is part of a septicæmia. The *symptoms* are those of acute meningitis; the C.S.F. is purulent. The organism causing the disease is found on culturing the C.S.F.; treatment depends on the organism and its sensitivities. The following is an *indication only* of the type of chemotherapy used in such cases:

Streptococcal meningitis: sulphonamides and intramuscular penicillin.

Pneumococcal meningitis: sulphonamides and penicillin (intrathecal and intramuscular), sometimes chloramphenicol.

Hæmophilus influenzae meningitis: intramuscular streptomycin + chloramphenicol.

Staphylococcal meningitis: penicillin (intrathecal and intramuscular) + chloramphenicol.

(c) Aseptic Meningitis

The symptoms are those of acute meningitis (usually rather mild). The C.S.F. is *clear*; cells and protein are raised but sugar is normal.

Aseptic meningitis is generally caused by a virus—e.g. that of mumps, or glandular fever; there are several other viruses which may attack the brain or meninges, one of which is called the *Coxsackie* virus. These viruses can be found in the C.S.F., but virus studies take a long time and do not influence treatment, which is symptomatic in all these cases; the viruses do not respond to chemotherapy. Complete recovery is the rule.

Non-paralytic poliomyelitis and *tuberculous meningitis* often resemble aseptic meningitis very closely. It is obviously particularly important to distinguish tuberculous meningitis, where delay in starting treatment may be fatal. The diagnosis is made on examination of the C.S.F.

(d) **Syphilitic Meningitis** has already been described (p. 294).

(4) **Diphtheria**

The successful campaign against this grave infection has been one of the greatest triumphs of preventive medicine. For many years diphtheria was the chief killer of school-age children, and there were some 60,000 cases yearly, with about 2,000 deaths. Diphtheria immunisation had long been known of, but little practised. In 1940 the Ministry of Health launched an immunisation campaign. About two-thirds of all children between one and fifteen have now been immunised, with the result that, in notifications had dropped to 37 with 6 deaths.*

Diphtheria is an infection contracted by inhaling the bacilli passed on by case or carrier. Both convalescents and people who have never had the disease may carry the germs in their throats. Sucking infected pencils or toys may also cause the disease. An attack usually conveys lifelong immunity.

Symptoms.—The incubation is short—about one to four days as a rule. The illness starts insidiously, especially in children. The child seems quiet, off colour and feverish; he does not eat, he may be sick once or twice and older children complain of a sore throat. If you look at the throat at this stage you will see a patch of *membrane* on one or both tonsils, the palate or back of the throat. The membrane is a shiny, greyish white; it has a well-defined, raised edge and slight bleeding occurs if it is dislodged with a spatula. The diagnosis is confirmed by taking a *throat swab* (p. 416) on which diphtheria bacilli will usually be found.

In grave cases the membrane may spread all over the back of the throat, up into the nasal passages and down into the larynx. If the

*In 1959 there were 102 notifications but—for the first time—no deaths.

larynx is involved breathing becomes difficult; the child draws in each breath with a crowing noise and the chest muscles are sucked in with the effort. In any severe case of diphtheria the glands of the neck enlarge, the face is puffy and ashen grey; the child lies sleepless yet apathetic. The pulse becomes feeble and irregular, the breathing rapid and shallow; urine is scanty and albuminous. *Vomiting* is often a herald of death, which in such cases occurs after a week or more of illness.

Children who escape or survive these grave symptoms may gradually recover, or they may develop one of the complications of diphtheria. During the third to fifth weeks the patient's *voice may become nasal* and when he eats *food regurgitates* through the nose. Examination of the throat shows that these troubles are due to *paralysis of the palate*. During the fourth to sixth weeks children who can read may complain that the print looks blurred; this is due to *paralysis of accommodation* (the mechanism whereby our eyes are adapted to looking at near objects). Finally, during the fifth to eighth weeks, when he gets up, he may find that he has lost the use of his limbs, so that he falls about and drops things—he has diphtheritic *polyneuritis* (see p. 306). Heart failure may occur at this stage. Diphtheritic polyneuritis may affect the breathing muscles, and the child may suffocate unless some form of mechanical artificial respiration is at hand.

Other Varieties of Diphtheria.—*Nasal diphtheria* may cause a blood-stained nasal discharge in a child who does not seem at all ill. Yet this child is a great danger to his associates, who are just as likely to catch severe diphtheria from him as from the more obvious case.

Laryngeal diphtheria may occur without obvious throat infection. Any child with a croupy cough and wheezing inspiration is suspect. A throat swab is usually positive for diphtheria bacilli.

The Cause of the Symptoms.—In diphtheria bacilli lodge and multiply in the throat and its neighbourhood; they do not spread all over the body as in typhoid, but they send out great quantities of *toxin*, and this poison causes most of the severe symptoms of the disease. The *membrane* is the result of the local infection; it consists of dead tissue mixed with diphtheria bacilli and exudate. The *toxin* poisons the heart and blood vessels, causing failure of the circulation—hence the feeble, irregular pulse and ashen pallor. Diphtheria toxin is also a nerve poison, which may cause paralysis of palate and accommodation or polyneuritis. (We have already seen [p. 306] that poisons which affect the nerves often affect the heart too.)

Treatment.—Diphtheria patients need the most skilful nursing. The patient is isolated and nursed flat, or with one low pillow, because of the danger of circulatory failure; he is kept flat for two to four weeks, or longer if the pulse is feeble or irregular. If, when he gets up, he is found to have polyneuritis he is put back to bed, to rest both damaged nerves and heart.

A liquid diet is easiest to swallow. If the palate is paralysed tube feeding may be necessary.

If there is circulatory collapse the foot of the bed is raised; adrenaline may be injected and glucose saline infusion given intravenously; intravenous hydrocortisone is often given too.

Antitoxin.—Diphtheria toxin may be counteracted by *diphtheria antitoxin*. Antitoxin is prepared from the serum of horses which have been immunised against diphtheria. Eight thousand to twenty thousand units intramuscularly are given in mild and moderate cases; much bigger doses can be given in severe cases. Antitoxin is always given if diphtheria is *suspected*, without waiting for the report on the throat swabs.

Treatment of Laryngeal Diphtheria.—If diphtheritic membrane in the larynx is choking the patient *tracheotomy* or *intubation* may save life by preserving an airway. In *tracheotomy* the trachea is incised and a tube inserted; *intubation* means passing a tube into the larynx from above. Usually the tube can be withdrawn or the tracheotomy allowed to close after three or four days, when the danger of asphyxia is generally over.

The outlook in diphtheria.—This is good in mild cases. In severe cases the outlook depends more than in almost any other disease on the medical and nursing treatment. With good nursing and prompt antitoxin treatment it is fair; otherwise it is grave.

Prevention of diphtheria.—Cases should be isolated for 4 weeks or until the throat swab is negative. Quarantine for susceptible contacts is 10 days. Contacts should be given small doses of anti-toxin, which will protect them from the disease for two or three weeks; this is an example of *passive immunity*.

Immunisation.—Every child should be immunised against diphtheria at the age of 8–12 months or earlier. Two injections of a modified toxin preparation are given a month apart; another injection is given at the age of 5 years, on entering school. These injections of toxin stimulate the body to produce *antitoxin*, so that if diphtheria infection later occurs it will probably have no effect.

By the time we reach adult life most of us are immune to diphtheria even if we have never had the disease or the inoculations. Those

who are still susceptible should be immunised if they are in contact with diphtheria—e.g. nurses in fever hospitals. *Susceptibles* are detected by means of the *Schick test*; a tiny dose of toxin is injected into the skin of the forearm and if a red wheal one-half to one inch across develops in 24–48 hours the person is *Schick positive and susceptible to diphtheria*.

(5) Scarlet Fever (Scarlatina)

Once the most dreaded of childhood's fevers, scarlet fever is to-day not very common, very severe or very infectious. Scarlet fever is a throat infection caused by *Group A hæmolytic streptococcus*—the same organism as causes acute tonsillitis (p. 354); it is, in fact, tonsillitis with a rash. The disease is usually contracted by inhaling the organisms, which may originate from a person with scarlet fever, tonsillitis or other streptococcal illness, or from a healthy carrier; occasionally *infected milk* is the source.

Symptoms.—The incubation period is about three days. The illness starts suddenly with vomiting, headache and sore throat. The patient is hot, dry-skinned and bright-eyed; the tongue white with red spots (strawberry tongue) and the throat red. The temperature rises rapidly to 103° F. or so, and the pulse rate may be 120 or more—up to 160 in children.

Within 24 hours a *rash* appears, consisting of little red dots crowded together over the chest and root of the neck, spreading later over the limbs. The face is not spotty, but the cheeks are brightly flushed while the skin round the mouth is notably pale (circumoral pallor). The *throat* is acutely inflamed and the tonsils may be covered with white exudate.

The patient usually begins to recover in a few days, and by the end of the week the temperature is normal, throat clear, rash faded and the skin is peeling. Peeling begins where the rash first appeared—over the chest and neck. Little pin-points of skin begin to peel off, and these tiny holes finally enlarge and merge together. The tongue peels; too, and looks shiny and red (raspberry tongue).

In the rare, severe cases of scarlet fever extensive throat sepsis, hæmorrhagic rash or septicæmia may occur, and such cases may be fatal. The general mortality of scarlet fever is less than one in a thousand.

The Cause of the Symptoms.—Scarlet fever, like diphtheria, is caused by organisms which lodge and multiply in the throat and send their *toxins* all over the body, causing the rash and other symptoms.

Complications. *Otitis media* (inflammation of the middle ear),

stomatitis (p. 353), and *cervical adenitis*, are all common; they are caused by the spread of organisms from the throat to neighbouring structures. *Nephritis* (p. 190) is not rare in the third week and *acute rheumatism* (p. 57) may occur from the second week onwards.

Treatment.—The usual nursing treatment of an infectious, febrile patient is necessary. The mouth and throat need regular cleansing. The urine must be tested daily for albumin. Albuminuria is common during the acute stage of any fever and is then not important, but albuminuria during convalescence may be a sign of nephritis.

Penicillin and *sulphonamides* are often given, especially if there are complications such as otitis media.

Prevention of Scarlet Fever.—Scarlet fever patients are usually isolated for three weeks. To prevent the spread of infection the patient should not be discharged if he still has a running ear or nose—such discharges teem with streptococci. The peeling skin is *not* a source of infection.

Quarantine for susceptible contacts is ten days.

(6) Erysipelas

Here is yet another possible result of infection with the versatile streptococcus. It is a skin infection; the bacteria usually enter through a small scratch or sore.

The incubation period is 2-5 days. The illness starts abruptly, often with a rigor; the temperature rises quickly to 102-103° F. and the patient suffers from headache and general discomfort. The affected area of skin feels hot and uncomfortable and looks red, shiny and slightly swollen; the well-defined margin of the rash is slightly raised and here a few tiny vesicles may be seen. Any part of the body may be attacked, but erysipelas of the face is especially common.

Treatment.—The patient should be isolated and nursed as for any febrile illness. Penicillin and/or sulphonamides rapidly cure nearly all cases of erysipelas.

(7) Whooping Cough (Pertussis)

This disease is one of the more serious of the common fevers of childhood. It is much harder to prevent than diphtheria, much more dangerous than scarlet fever and much commoner than either. It is apt to attack the very young, and even new-born babies may get it, whereas most other infectious fevers are uncommon in young infants; the younger the patient the worse the disease.

Whooping cough is usually caught from a patient in the early stages of the disease; it is doubtful if clothes, toys, etc., or a healthy carrier can spread the infection. The responsible bacilli are expelled in infected droplets when the patient coughs; the next victim is infected when he inhales these droplets and the bacilli attack his air passages. One attack practically always gives lifelong immunity.

Symptoms.—The incubation period is about a fortnight. The disease starts gradually with a cough and, sometimes, fever, but there is no whoop in the early stages. In this *catarrhal* stage the disease is at its most infectious, and, as it is unlikely to be diagnosed

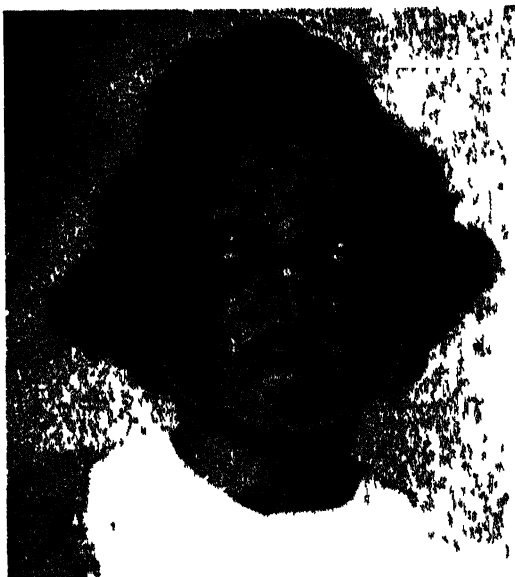


Fig 89. Whooping Cough
Note sub conjunctival hæmorrhage caused by the strain of coughing
(From Fanconi and Wallgren's Textbook of Paediatrics Heinemann)

the child is allowed to go about infecting his playmates. If, however, whooping cough is suspected, because the child has been in contact with a case, a *post-nasal swab* may reveal the responsible organisms. After a week or two the cough gets worse and comes on in *paroxysms* which are *worse at night*; a coughing fit makes the child *red in the face* and often causes *vomiting*; these four signs make the diagnosis of whooping cough likely even before the whoop develops.

Sometimes the child with pertussis never whoops at all; this makes diagnosis very different.

In the *paroxysmal stage* the diagnosis is obvious. Several times a day the child is seized by a fit of coughing which makes him start up in bed or run in terror to his mother. First comes a group of short, bark-like coughs, then a pause, during which the child's face becomes purple, then suddenly the air is drawn in with a long crowing whoop. This may be repeated several times before the paroxysm is over, when the child often vomits. Paroxysms are worse and more frequent by night.

The paroxysmal stage lasts some three to ten weeks, after which the cough gradually abates. In severe cases *bronchopneumonia* develops and may be fatal or may lead on to bronchiectasis. *Convulsions*, followed in some cases by coma or paralysis, are a rare but serious complication. The strain of the cough may cause *hæmorrhages* from nose or mouth, or under the conjunctiva or skin. *Heart failure* sometimes occurs.

Treatment.—The patient must be isolated, but he can be up and about and out of doors when the temperature is normal. He should eat nourishing and appetising food. If he is repeatedly sick he should be given something to eat just after vomiting.

Linctuses are given for the cough (p. 130). Belladonna or allied drugs such as atropine methonitrate ("eumydrin") may relieve the spasms. Chloramphenicol or tetracyclines, if given during the early catarrhal stage are sometimes thought to cut short the disease; given later they are useless.

Prevention of Whooping Cough.—Cases are usually isolated for four weeks. Quarantine for susceptible contacts (i.e. those who have not had the disease) is three weeks.

Whooping Cough vaccine may be given in infancy, often together with diphtheria immunisation and, in many cases, tetanus toxoid. ("Triple antigen" contains diphtheria and tetanus toxoids and whooping cough vaccine.) The vaccine should be given at about the age of three months if it is to prevent whooping cough in young infants to whom it is most dangerous. Three injections are necessary at monthly intervals.

(8) Measles

Like whooping cough, measles is extremely common, extremely infectious, and may be dangerous in the very young and badly nourished. Again like whooping cough, measles is nearly always caught direct from a case by *droplet infection*. Finally, whooping cough and measles are both diseases which nearly everyone has once, but once only—second attacks are very rare. People who say they

have had measles twice have usually had measles and rubella (p. 336).

Epidemics of measles occur pretty regularly every second or third year, usually in the late winter and early spring.

Unlike whooping cough measles spares the new-born. This is because the baby is born with its mother's immunity, if she has ever had measles, but this immunity fades after six months. Measles is caused by a *virus* which attacks the air passages and spreads all round the body, causing the rash.

Symptoms.—The incubation period is usually nine to twelve days. The first symptoms are those of a *bad cold*. The child coughs and sneezes with streaming eyes and a woebegone appearance. The temperature rises quickly to about 103° . *It is most important to recognise measles at this stage* because it is now at its most infectious. The rash has not yet appeared, but if you look in the mouth with a good light (preferably daylight) you will see groups of white dots, like grains of salt upon a red base, inside the cheeks opposite the back teeth. These are *Koplik's spots* and are a certain sign of measles.

The rash generally comes out on the fourth day of the disease, and just before this the temperature may fall, to rise again steeply as the spots appear. The spots are deep pink, slightly raised blotches, which are first seen behind the ears, on the forehead and round the mouth, and soon spread all over the body. The temperature is high, the breathing quick and shallow; there is a rasping cough, the eyes and nose run and the child is most miserable. In straightforward cases the rash and other symptoms clear up after a few days and the child is soon better. As the spots fade they leave a faint brown stain; this, too, fades in a matter of days.

Complications.—Measles attacks all the air passages, and there is always some laryngitis, tracheitis and bronchitis. The two commonest complications are *bronchopneumonia* and *otitis media*. Both respond rapidly to modern chemotherapy but both may have serious results; if neglected, bronchopneumonia may lead on to bronchiectasis, while otitis media may become chronic, causing deafness. A mild *stomatitis* occurs in all cases of measles, but in severe cases the mouth is ulcerated and foul and *noma* (gangrenous stomatitis) may occur; fortunately this terrible complication is rare (p. 354). *Encephalomyelitis* (p. 298) is another rare but serious complication.

There is no specific treatment of measles. Bed rest, isolation and the usual nursing treatment of feverish patients are necessary. The *mouth* must be carefully cleansed daily; if the *eyes* are inflamed they should be bathed with warm saline and golden eye ointment applied to the lids. A soothing *linctus* is given for the cough. In uncom-

plicated cases the child may get up two or three days after the temperature has fallen, and should then have as much fresh air and good food as possible. *Complications* are treated as they arise.

Prevention of Measles.—Patients should be isolated until the temperature has been normal for three days. The patient is most dangerous during the few days of sneezing and coughing before the rash is out. Suspect any child with a feverish cold if there is measles about, and look for Koplik's spots. Quarantine for susceptible contacts is sixteen days; as nearly everyone gets measles sooner or later it is, in this writer's opinion, wasteful to make *older children* lose precious weeks of school or holidays in observing precautions which only postpone the inevitable.

Measles can be prevented or made less severe by giving an injection of *gammaglobulin* during the incubation period. Gamma globulin is a preparation, made from pooled human plasma from many donors, which contains antibodies against measles and other infections the donors may have had.

Gammaglobulin is scarce and expensive. It is usually reserved, therefore, for those in whom an unmodified attack of measles might be dangerous—very young children, or those already suffering from some other illness. It may also be used to prevent an outbreak of measles in a children's ward should a child develop the disease soon after admission.

A measles contact who receives a large dose early in the incubation period will not develop the disease. This is called *passive immunisation*, because the child passively receives the immunity to measles possessed by the donors of the blood plasma. Passive immunity wears off in three or four weeks, leaving the child as susceptible as ever. So it is usually better to give *less* gammaglobulin, or to give it *late* in the incubation period (between the fifth and ninth days); the child will then have a very mild attack of measles, after which he will have lifelong *active immunity*, just as he would after an unmodified attack.

(9) Rubella (German Measles)

Rubella, mumps and chickenpox, unlike the diseases so far described, seldom cause serious illness, but they have a high nuisance value on account of their long quarantine periods. Rubella is the mildest of them all; it has an incubation period of 16–18 days. It is important for only two reasons—it may be confused with measles or scarlet fever, and if it attacks a woman during the first three months of pregnancy the baby is likely to be born deaf or with congenital cataract or heart disease.

Rubella is caused by a virus. Infection is usually direct from patient to patient by droplet spray; the disease is not highly infectious. One attack generally protects for life.

Symptoms.—The rash may appear without warning, or there may be a day or two of catarrhal symptoms first. The spots are smaller and paler than those of measles; as in measles they appear first on the face and neck. After twenty-four hours the spots may run together, mimicking scarlet fever. There are no Koplik's spots, no severe throat infection, no peeling and no staining. *The glands at the back of the neck are tender and enlarged to the size of peas*—a very characteristic sign. The glands may be enlarged before the spots appear. The temperature may be raised for a day or two; usually the whole illness is trifling and quickly over.

No special treatment is necessary; the patient may hardly need to go to bed.

Prevention.—The patient should be isolated till symptoms have subsided. Quarantine, where this is enforced, is 21 days, but it does not seem right to make a child lose three weeks' school or holidays to avoid such a trifling disease. Besides, girls should be glad to get it over, to avoid the later possibility of rubella during pregnancy and a deaf baby. Of course, a woman in the early stages of pregnancy should do everything to avoid catching rubella. If it is known that such a woman has been exposed to rubella she should be given gammaglobulin; it is said to prevent rubella as it does measles.

(10) Mumps (Epidemic Parotitis)

This disease, only moderately infectious, is caused by a virus; infection is direct from case to case by droplet spray. One attack generally conveys lifelong immunity.

Symptoms.—The incubation period is about three weeks. The first symptom is usually a swelling at the side of the face, which fills up the groove just behind the jaw and lifts the ear away from the head. This region is the site of the *parotid gland*—the largest of the three pairs of salivary glands. The submaxillary and sublingual salivary glands may be affected too, causing swelling under the chin. As a rule one gland is affected first and the other after a day or two. There may be a slight rise of temperature as each gland enlarges.

When both glands are enlarged eating is difficult and painful, as the patient can hardly open his mouth or chew. The pain is worse during eating because the glands then produce more saliva, which increases the swelling. The swelling subsides after some days.

Complications.—The most important is *orchitis* (inflammation of the testicles) which occurs in 15-20 per cent. of adolescent boys and young men with mumps, about the seventh or eighth day of the disease. The testicles become enlarged, hot and tender, and the patient is feverish. Luckily the disease usually clears up completely, but *sterility* occasionally results.

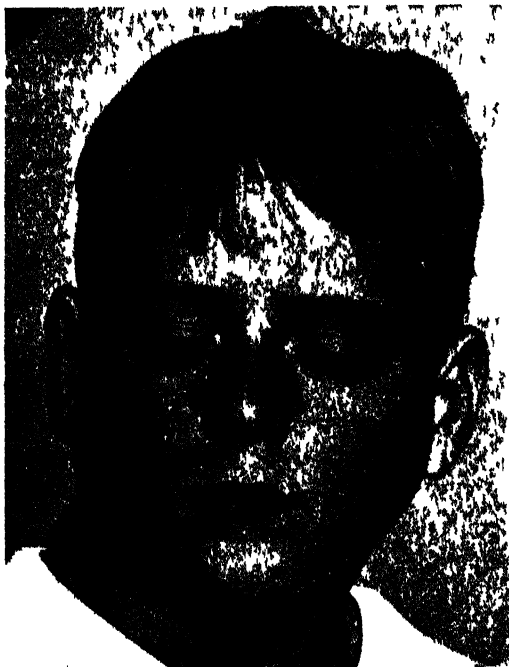


Fig. 90 Mumps
(From Fanconi and Wallgren's Textbook of Paediatrics Heinemann)

Encephalomyelitis (p. 298) may occur between the seventh and tenth days. *Pancreatitis* may simulate an abdominal emergency. Other rare complications are nerve deafness, pericarditis, nephritis and optic atrophy.

Treatment.—There is no special treatment; bed rest until the danger of orchitis is passed is desirable for male patients. The patient should have a fluid diet while chewing is difficult. Complications are treated as they arise.

Prevention.—Patients should be isolated for two weeks from the beginning of the illness. Quarantine for susceptible contacts is 28 days. Adolescent boys and young men should try to avoid the

disease, but in girls it is seldom serious and in small boys it should be welcomed because they will then be immune to mumps when the danger of orchitis is greatest.

(11) Chickenpox

This is a highly infectious disease caused by a virus. Like measles it is almost impossible to avoid catching it at some time or another;



Fig 91. The rash in Chickenpox
Note that the spots are at different stages of development.
(From Fanconi and Wallgren's *Textbook of Paediatrics*, Heinemann)

second attacks are exceedingly rare. The infection is generally passed on direct from case to case by droplet spray, but the scabs are infectious and may be carried on clothes, etc.

Symptoms.—The incubation period is about a fortnight. The rash is generally the first sign of the disease, though adults may have some fever and headache for a day or two first.

The spots appear first on the trunk, then on the face, scalp, axilla, groin, and upper parts of the limbs. Spots may be seen in the mouth

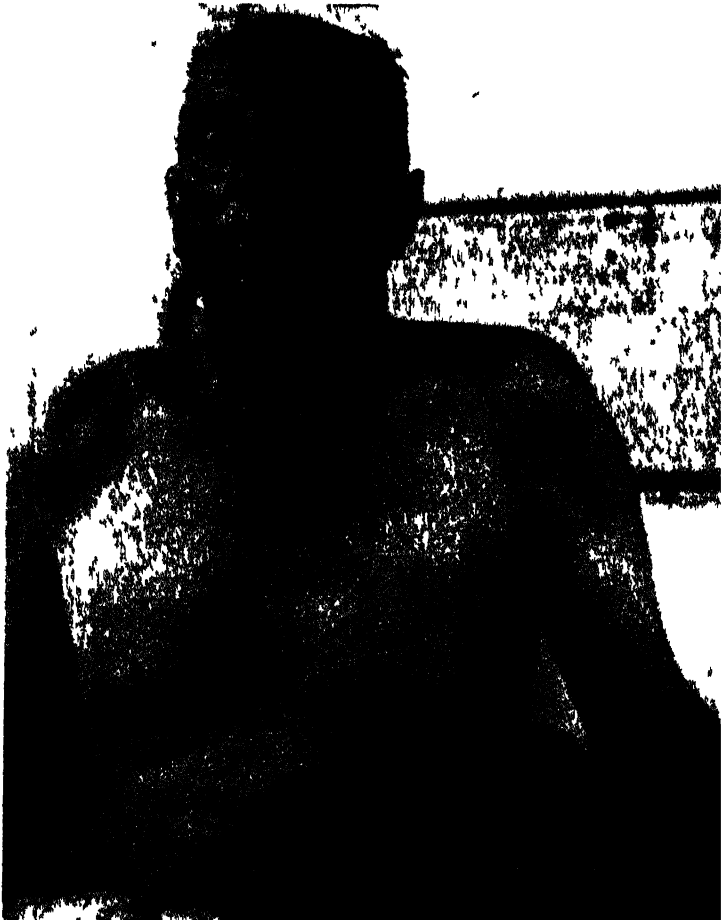


Fig 92 Shingles and Chickenpox

A rare combination This man has shingles of the upper part of the face (ophthalmic zoster) (Dr Richard Asher's case)

and throat. Successive crops appear over the next few days. Each crop consists of a number of pink spots, flat at first but soon becoming raised (*papular stage*). Clear fluid collects in the papules forming tiny blisters or *vesicles*, and this fluid at length becomes turbid.

forming *pustules*. A spot becomes a pustule in about twenty-four hours. Within a day or two the pustules dry up and form scabs, which fall off leaving pink marks but no obvious pits. Slight fever usually accompanies the rash.

The spots are very itchy and scratching may lead to secondary sepsis. Serious complications are rare.

Treatment.—There is no specific treatment. The patient need not be kept in bed unless he is feverish. Dusting powders and calamine lotion may allay itching; secondary sepsis may call for sulphonamides or penicillin.

Prevention.—The patient should be isolated till all scabs have separated. Quarantine for susceptible contacts is three weeks.

(12) Smallpox (*Variola*)

Smallpox is rare in this country, though small outbreaks occur from time to time when the infection is brought in from abroad. It is one of the most infectious diseases known. Like chickenpox, it is caused by a virus which is usually spread by droplet spray, but may also be found in the crusts and pustular fluid in the spots. (In one outbreak several cases occurred among workers in a laundry to which was sent the linen of an undiagnosed case of smallpox.) Second attacks of smallpox are very rare.

Symptoms.—The incubation period is ten to fourteen days. In major smallpox (*variola major*) the patient is suddenly smitten with headache, pains in the loins and rigor, and he rapidly becomes very ill. The temperature shoots up to about 104. There may be a fleeting rash like that of scarlet fever or measles but the true smallpox rash does not appear till the *third day*.

The smallpox rash is first seen and is always worse on the *face, hands and feet*—contrast chickenpox, where the *trunk* is chiefly affected. The rash consists of pink spots which in five days develop through papule and vesicle to pustule *in unison*, not in a series of *crops* as in chickenpox. On the face and hands the spots may be so numerous that they run together. The temperature, which falls when the rash first appears, goes up again when the spots become pustular, and the patient is very ill. After the sixth day the rash begins to dry up, and finally the temperature falls and scabs separate leaving deep pits or *pocks* behind. The mortality of major smallpox is 25–50 per cent.

Mild smallpox (*variola minor*, *alastrim*) is a much milder illness which may easily be confused with chickenpox. It may be caused by

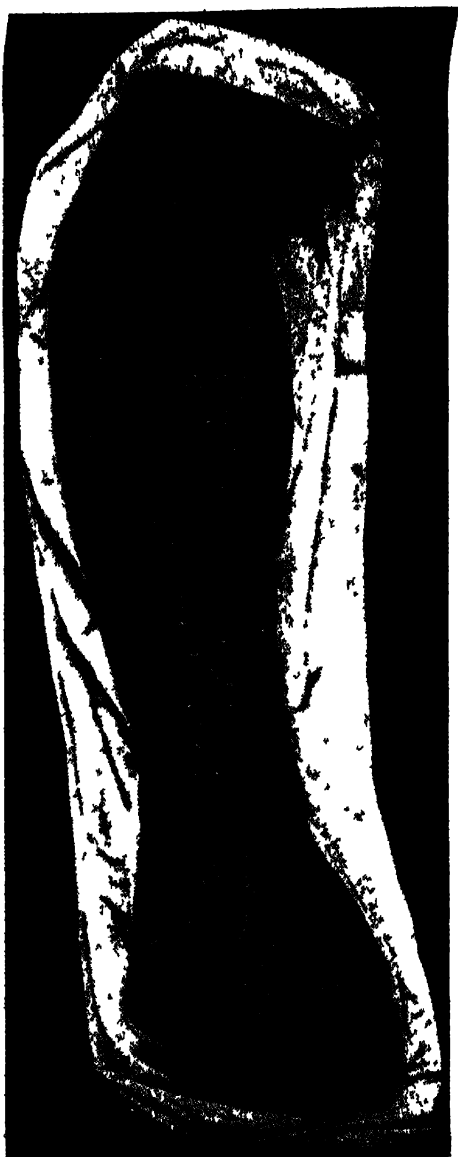


Fig. 93. Smallpox
Pustular stage of rash. Note that lesions are practically all at the same stage of development.
(From Jacobi's *Atlas of Dermochromes*, Heine-
mann.)

a different variety of virus, or by the major infection in a vaccinated person.

Treatment.—There is no specific treatment; good nursing is essential. Sedative and pain-relieving drugs are given as required; high fever calls for tepid sponging. The rash is treated with potassium permanganate or dilute carbolic acid solutions. The mouth must be regularly cleansed. Antibiotics help to combat infection during the pustular stage.

Prevention.—Smallpox cases must be rigorously isolated till all scabs have separated; everything used by the patient must be sterilised. All known contacts should be vaccinated. The quarantine period is sixteen days.

Vaccination gives almost certain protection against smallpox for about two years, and will probably modify an attack for many more years. In vaccination we infect a person with a *living virus*—that of vaccinia, or cowpox, an infection closely related to smallpox, but very much milder. This mild infection gives the person *active immunity* against the much graver disease. It should be remembered, however, that a successful vaccination is a mild *disease*, not just the injection of dead bacteria as is a typhoid inoculation.

Vaccination has been practised on a large scale in most civilised countries since it was first described by Edward Jenner in 1796. It has enormously reduced the mortality from a disease which was once as common in this country as measles is to-day.

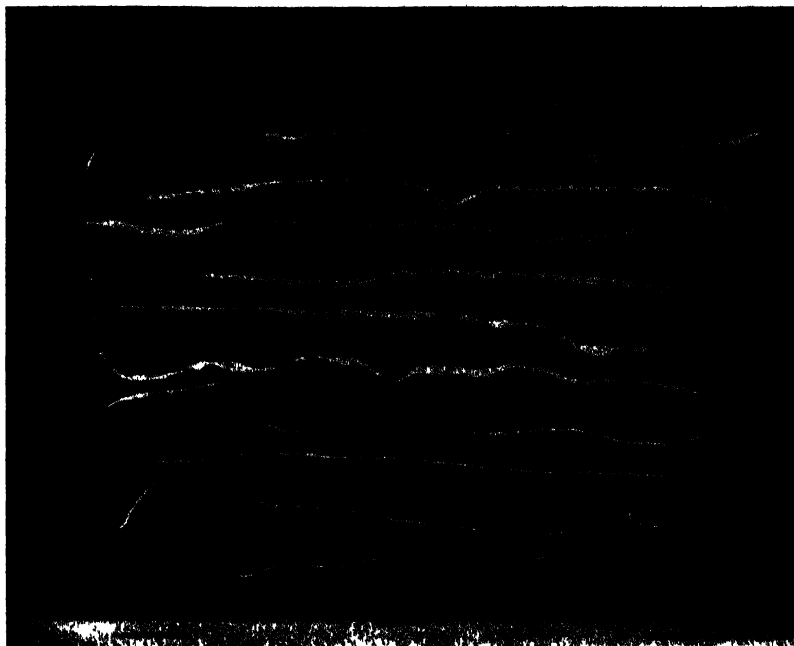
The vaccinia virus is obtained from calves infected with cowpox. Lymph from these calves is specially treated and put up in glass tubes; the lymph must be used within seven days unless kept in a refrigerator.

Procedure.—The skin is cleansed with ether or soap and water and dried with a sterile swab. A drop of lymph is placed on the skin. A scratch $\frac{1}{4}$ inch long is made through the drop of lymph, which is then allowed to dry; the scratch must not be so deep as to draw blood. A sterile dressing is then applied. If the vaccination is successful a pink papule appears on the third day, which develops into a vesicle on the fifth and an itchy pustule on the eighth day. The tissues round the pustule may be very swollen, red and painful, and the temperature may rise, especially when adults are vaccinated for the first time. After a few days the pustule begins to dry and a scab finally separates leaving a pitted scar. If the vaccination does not "take" it should be repeated.

Like many other virus diseases (e.g. measles, mumps) vaccinia may be complicated by encephalomyelitis (p. 298); fortunately this

grave disease is rare. It is commonest when children aged six to fourteen are vaccinated for the first time.

Vaccination is usually recommended during infancy; although smallpox is now so uncommon, occasional outbreaks do occur necessitating vaccination of numerous contacts. Adults who are vaccinated for the first time are apt to have severer reactions than babies; even many years after the original vaccination a second vaccination usually causes little disturbance. Babies with infantile eczema, impetigo, etc. however, should *not* be vaccinated. This is because the cowpox virus may spread widely causing *generalised vaccinia* which is occasionally fatal. Nor should the contacts of a baby with eczema be vaccinated; the baby may catch generalized vaccinia from the vaccinated person.



(1)



(2)

Fig. 94. Beef Tapeworm (*Taenia Saginata*).
Expelled worm, with foot rule to give idea of the size. The thin end is the head end. 2. Head, highly magnified. (Dr. Richard Asher's case.)

(b) Tapeworms

These monstrous parasites spend part of their lives in man's gut, part in the flesh of pig or cow. The *beef tapeworm* (*Tænia saginata*)—the commoner variety in this country—is four or five yards long and a quarter of an inch wide. The adult worm lives in the gut, its head fixed to the intestinal wall by means of suckers. The worm lays ova which develop into embryos; these pass out in the fæces and may then be eaten by a cow. The embryos work their way through to the animal's flesh and form "cysts" in the muscles. If this meat is eaten, somewhat underdone, the cysts develop into worms in the eater's intestine.

Symptoms.—Curiously enough it is possible to harbour several yards of tapeworm and be quite unaware of the fact. Sometimes, however, the patient may have diarrhœa, colic or bouts of voracious appetite.

Treatment.—First of all the patient is given a fluid diet and dosed with Epsom salts for two days. This makes the stools fluid, and several yards of tapeworm are dislodged in the process. However, unless the *head* is dislodged the worm will grow again. The drug for this is *extract of male fern* (*aspidium filix mas*). On the third morning three to eight capsules are given to the fasting patient at fifteen-minute intervals; one hour after the last dose half an ounce of Epsom salts is given. During the course of the day every stool must be carefully searched for the head of the tapeworm; the liquid stools make it easier to find. The tapeworm gets narrower and narrower nearer the head, till it is as thin as a thread; the head will be seen at the end of this thread as a tiny round dot, the size of a full stop. The treatment, if unsuccessful, is repeated in ten day's time. *Mepacrine* may be used instead of *filix mas*, a single dose of 0.5 gm. being given first thing in the morning. The rest of the treatment is the same.

Cysticercosis.—If a person swallows pork tapeworm embryos the *cyst* stage of the worm may develop in his body. The cysts may be felt as little nodules under the skin or in the muscles. Eventually the cysts become calcified and show up on X-ray. Cysts in the brain may cause epilepsy. No curative treatment is known.



Fig 95 Cysticercosis
X-rays (of abdomen and legs) showing pork tapeworm cysts in muscle. The patient had epilepsy, caused by cysts in the brain (Dr Porter's case)

(c) Roundworms

These look something like earthworms. If a person swallows the ova larvæ develop which migrate all over the body before settling down to develop into adult worms. Often there are no symptoms other than the appearance of worms in the fæces.

Treatment is with oil of chenopodium, carbon tetrachloride or piperazine preparations. The worms are ejected in 48 hours. The person is cured if no more ova are found in his stools.

DISEASES OF THE NOSE, MOUTH AND THROAT

(1) Hay Fever

(Paroxysmal Rhinorrhœa, Allergic Rhinitis)

THIS disorder is common in families who suffer from migraine, asthma, eczema or urticaria. As in other allergic diseases, the sufferer is sensitive to minute quantities of some substance which is harmless to the rest of us. In hay fever this substance is *plant pollen*; in other varieties of allergic rhinitis dust, face powder, horse hair or various foods may be incriminated.

In an attack of hay fever the patient sneezes uncontrollably, with streaming eyes and nose. If pollen is responsible the attacks come on between May and August.

Treatment.—Sometimes we can *prevent* hay fever by giving the patient a course of desensitising injections of pollen extracts, starting with a very minute dose; the injections should be given in winter and early spring. The antihistamine drugs (p. 269) are effective in many forms of allergic rhinitis. A course of tablets may ward off attacks; should an attack occur nasal drops containing one of these drugs may cut it short.

(2) Epistaxis (Nose Bleeding)

Nose bleeds are common in many diseases—high blood pressure, blood diseases, heart failure, and in most of the common infectious fevers. Many healthy people seem to bleed from the nose from very little cause. These people usually have a little patch of fragile blood vessels on the nasal septum, just inside the tip of the nose.

On no account should the patient lie down during a nose bleed; if he does the blood runs down the back of the throat to be coughed up or vomited later, much to the patient's alarm. Instead he should sit up, blow his nose to get rid of blood and clots, and compress the nostril firmly with his fingers (bleeding is practically always confined to one nostril). Pressure is kept up till bleeding has stopped—usually within a few minutes. Very occasionally bleeding is severe and prolonged and is not stopped by this simple manœuvre. The affected nostril may then have to be packed with ribbon gauze. The gauze must be removed within 24 hours.

If nose bleeds are very frequent the blood vessels which are the source of the bleeding may need cauterising.

(3) Laryngitis

The larynx, or voice box, is inflamed in many acute infections, such as the common cold, influenza, measles and whooping cough. Sometimes laryngitis follows straining the voice, as may occur at a political meeting in a large, smoky hall.

The chief symptom of laryngitis is huskiness; sometimes the patient can only whisper. He may also have a sore throat and a painful, dry cough.

In *children* laryngitis may be a serious disease, as in them the opening between the vocal cords is so tiny that it is easily blocked by secretion so that the breathing is obstructed. In these very young patients, too, the inflamed larynx is apt to go into spasm, thus adding to the difficulty of breathing. The child then draws in each breath with great difficulty, making a crowing noise as he does so.

Treatment is that of the cause, where this is known. Steam inhalations are often soothing, and linctuses or cough lozenges may ease the cough and sore throat. In young children with severe laryngitis a steam kettle is advisable. Sometimes an *emetic* (a drug which makes the child sick) relieves the spasm.

Laryngeal diphtheria is described on p. 329.

Laryngismus stridulus is a spasm of the larynx which may occur in unhealthy, rickety children with adenoids. The attacks come on as a rule at night and are very alarming. The child starts up gasping for breath and suddenly stops breathing altogether. He goes purple in the face, his eyes start from his head and, when he seems to be at the last gasp, he draws in his breath with a long drawn out crowing noise. The attack once over he seems quite well and not at all hoarse.

Treatment.—The tongue should be drawn forward by a finger passed into the back of the mouth; this usually stops the attack. Afterwards the child's general condition should be investigated and treated.

(4) Stomatitis (Inflammation of the Mouth)

Anyone suffering from an acute feverish illness may have an inflamed mouth. Stomatitis is particularly common in typhoid fever (p. 316), in measles (p. 334), and after any severe abdominal operation. If a person does not eat or drink much his salivary glands do not

secrete much saliva; if he lies unconscious or semi-conscious for many hours his mouth may drop open, when the mucous membrane soon dries. A dry, foul mouth in such patients can be prevented by good nursing; the mouth should be cleansed after every meal, or several times a day in unconscious patients.

Stomatitis also occurs in agranulocytosis (p. 154), in scurvy (p. 365) and in mercurial poisoning.

Apart from these serious illnesses, simple *catarrhal stomatitis* may result from over-smoking, ill-fitting dentures, etc., when the mouth is dry, red and uncomfortable.

Aphthous Stomatitis.—The cause of this very common condition is unknown. Little vesicles appear on the mucous membrane lining the cheeks, gums and other parts of the mouth. These vesicles soon rupture, leaving shallow yellowish-white painful ulcers. Many people are subject to aphthous stomatitis, the ulcers appearing singly or in crops at frequent intervals for years.

Treatment.—No curative treatment is known. A simple alkaline mouthwash is often soothing; hydrogen peroxide may be used if there is much secondary infection. No method of preventing recurrences is known either. Vitamins are ineffective.

Ultero-membranous stomatitis is an infectious disease caused by the same organism as Vincent's angina (p. 355). The margins of the gums are swollen and bleed easily, the teeth may loosen and the breath is foul. All parts of the mouth may be affected. The treatment is that of Vincent's angina, together with strict cleanliness of the teeth and mouth.

Gangrenous stomatitis (noma) is fortunately rare. It used sometimes to complicate measles and other acute fevers in debilitated children. Sloughing ulcers form in the mouth and much of the face may be destroyed. The disease is treated surgically; the death rate is very high.

Thrush.—This is common among ill, badly nourished, bottle-fed babies but may occur in others. The inside of the mouth is speckled with white spots; in severe cases ulceration occurs. The disease is caused by a yeastlike fungus. It is readily cured by painting the inside of the mouth with gentian violet (1 per cent. solution) twice daily for two weeks. Local applications of the fungicide *trystatin* are also effective.

(5) Acute Tonsillitis

Inflammation of the tonsils is common in childhood and early adult life, and causes *acute fever* and *sore throat*. Children do not

complain of sore throat as readily as adults, and one is often amazed to see a pair of large, inflamed tonsils on examining the throat of a child with a temperature but no other symptoms. The tonsils are red, swollen and speckled with yellow, purulent exudate which can easily be scraped off with a spatula (cf. diphtheria, p. 328). The glands in the neck, just behind and below the angle of the jaw, are swollen and tender.

Tonsillitis generally responds quickly to treatment with penicillin or one of the sulphonamides, together with the usual nursing treatment of a febrile illness. If a person has repeated attacks of tonsillitis his tonsils may have to be removed.

Tonsillitis is most often caused by the *hæmolytic streptococcus*. If the patient has a rash as well the disease is called scarlet fever, which is a notifiable infectious disease (p. 316). Tonsillitis is not usually treated as an infectious disease, but streptococci can be passed on just as well by these patients as by those with scarlet fever, and complications, such as otitis media, rheumatic fever and nephritis follow tonsillitis as often as scarlet fever. Nurses should impress on their patients that tonsillitis is infectious. Young children in particular should be kept away from tonsillitis patients. General practitioners encounter many family outbreaks of streptococcal infection. A child develops tonsillitis; a few days later another child develops otitis media. Finally the baby becomes ill with a high temperature for which there is no obvious cause. All three have an infectious illness caused by the same strain of streptococci yet none are isolated. On other occasions scarlet fever, erysipelas or impetigo may develop in tonsillitis contacts.

Enlarged tonsils are common among children and are not necessarily unhealthy. The tonsils may be large because they are zealously carrying out their proper function of entrapping bacteria which were about to enter the lungs. Tonsils should not be removed just because they look big.

(6) Vincent's Angina

This is a variety of tonsillitis caused by a pair of organisms—one long and one twisted—called *Vincent's organisms*. It comes on more gradually than the disease described above and the throat may not be very sore. Only one tonsil is affected as a rule; it is covered with a mass of crumbly, yellow membrane which leaves behind a clear-cut ulcer when it comes away. The breath is foul and the glands enlarged.

Vincent's organisms, like those of syphilis (another corkscrew

organism—see p. 346) respond to penicillin treatment. Dental treatment is necessary if the gums are affected, or relapses may occur.

(7) Quinsy

This is an abscess which forms just outside the tonsil. It causes an acute illness; the patient feels ill, the temperature shoots up, there may be a rigor; there is intense pain in the throat; the breath is foul and the corresponding glands are enlarged. The patient cannot swallow and can hardly open his mouth, so examination is difficult, but with a good light the quinsy can be seen bulging out one tonsil and distorting the soft palate.

Treatment.—Aspirin, pethidine or even morphia may be necessary for the pain. Large doses of penicillin by injection may be curative if given very early in the infection. In most cases, however, the abscess goes on growing until it bursts or is opened. The pus then discharges and the relief is instantaneous. The temperature falls and the patient rapidly gets better. Later, tonsillectomy may be necessary.

(8) Cardiospasm (Achalasia of the Cardia)

This is a disorder of the lower end of the œsophagus (gullet). The œsophagus is very rarely inflamed, and strictures and new growths of this organ are described in textbooks of surgery, so that cardiospasm is the only œsophageal disease we shall discuss.

In cardiospasm the patient says that the food sticks in the gullet. At first this happens for a day or two at long intervals, but as it gets worse the condition at length becomes permanent. After a meal there is a feeling of tightness in the chest and quite soon the whole meal may come back, without any feeling of nausea. The patient may waste away from lack of nourishment.

The *cause* of the trouble is in the sphincter which guards the entrance to the stomach. This should open as soon as food reaches the lower end of the œsophagus; it does not do so in cardiospasm and food collects in the œsophagus which then becomes dilated—it may have a capacity of 2 pints, whereas the normal œsophagus is but a narrow tube.

In the early stages symptoms may be relieved if the patient avoids bulky, irritating or very cold food and alcohol. *Nitrites* may relax spasm and allow food to pass. In severe cases surgical treatment is required.

(9) Dysphagia (difficulty in swallowing)

Cardiospasm is one cause of dysphagia. Other causes are obstruction of the œsophagus by growths, etc. Women with iron deficiency anæmia have difficulty in swallowing; so, of course, have patients with any variety of sore throat. Dysphagia occurs in diseases such as myastheniagravis (p. 304), and bulbar poliomyelitis (p. 292), because the muscles of swallowing are paralysed. Diphtheritic paralysis of the palate (p. 329) also makes swallowing difficult.

CHILDREN'S DISEASES

MANY illnesses have already been described which affect children as well as adults and, of course, most of the infectious fevers are commonest in childhood. In this chapter are described a few conditions which are practically confined to childhood. First of all, however, here are a few notes on the nursing of children and infants.

Children are not dwarf adults. Their needs differ from those of adults both in sickness and in health. Young children and babies differ from adults in their reactions to infection, to heat and cold, and to loneliness. Let us look first at children's reactions to infection. Many infections run quite a different course at different ages. For instance, *tuberculosis* (p. 108) is apt to cause miliary tuberculosis or meningitis in young children, but the chronic lung disease seen in adults is rare. *Streptococcal infection* may cause otitis media, pneumonia or septicæmia in an infant, but rarely the tonsillitis (p. 354) or scarlet fever (p. 331) so common among older children. The *common cold* is a nuisance to an adult, an affliction to a child of three, and may cause a fatal illness in a premature baby.

In general, the younger the patient the more susceptible he is to infection, for the body's defence mechanisms are not fully developed at birth. Because of this, and because of the risk of cross infection, babies are only admitted to hospital when it is absolutely necessary, and barrier nursing is often advised even in "clean" infants' wards. (Some of the infectious fevers, however—measles, chickenpox, mumps, for instance—are rare under the age of 6 months, because the babies are born with their mothers' immunity to these infections.)

Acute infections in infancy, especially when there is high fever, often cause *convulsions*. A convulsion may be the first symptom of almost any infection—e.g. pyelo-nephritis, otitis media or pneumonia. If an adult has a fit we think of epilepsy or a disease of the central nervous system, and neurological investigations follow. If a baby has a fit we turn first to the thermometer and the auriscope, though a lumbar puncture may sometimes be necessary. The most important part of the treatment is that of the infection, though an anti-convulsant such as phenobarbitone is given too.

Young children develop *high temperatures* much more often than

adults. A temperature of 104 or 105° is quite common in many infections, and is not necessarily a grave sign. However, if the temperature goes up to 106 or 107 this is a grave sign. Hyperpyrexia, as it is called, often develops unexpectedly, but it is a *particular danger when children are nursed in oxygen tents*, because of the heat and humidity inside these tents. If an obviously ill child is unusually restless and feels hot to the touch *take his temperature immediately*—it may have gone up several degrees in the last hour or two. A four-hourly temperature chart cannot be relied on to diagnose hyperpyrexia promptly.

A sudden dangerous rise in temperature must be immediately reported to the doctor. Tepid sponging (p. 20) will probably be called for. The child should be allowed to lie naked on his bed until the desired fall in temperature has occurred—this will not occur if he is immediately piled with blankets after the sponging. If he is having oxygen his head may be enclosed in the oxygen tent while the rest of his body is exposed. Special measures to diagnose and treat the cause of the hyperpyrexia may also be necessary.

Babies—especially newborn babies—are also very susceptible to *cold*. It has only recently been realised that normal, healthy newborn babies can be killed by cold. The condition is called *cold injury of the newborn*. A baby born at home during very cold weather may easily become chilled, especially at night, when the bedroom fire is allowed to go out. The baby looks pink and cries little, so that at first no one suspects that anything is wrong. Later he becomes more and more lethargic, lies motionless and refuses his feeds. His temperature, taken with a special low-recording thermometer, is between 80° and 90° . Such a baby may die of respiratory failure within a few hours unless re-warmed. Naturally, cold injury is rarely seen in centrally heated hospitals (though it does occur) but nurses should be aware of the danger of cold to young infants. Babies should not be exposed to temperatures of less than 65° for the first few weeks of life; arctic bedrooms with open windows may be all right for adults but are dangerous for the newborn.

Thus we see that young children are more susceptible than adults both to infection and to changes in temperature. They are also much more likely to be upset by admission to hospital and separation from their families—particularly the mother. The situation cannot be explained to a child of one or two, who may feel terrified, deserted or resentful. While in hospital he may win the golden opinion of the nursing staff for his angelic behaviour—but beware! It is quite unnatural for a child of two to sit or lie quietly in a cot, and such

behaviour may mask much unhappiness. Once home, the mask is dropped, and the child may scream, refuse his food or cling to his mother night and day.

To soften the blow of separation daily visiting is now encouraged in most children's wards, visiting at any time of day in many, and in some the mother is admitted with the child if she is able and willing to leave her home and the rest of the family.

These arrangements make fresh demands on the nursing staff, who have to spend a great deal of time answering queries, showing the mother what to do (if she is helping to look after the child) or even suggesting ways of passing the time. But many nurses find it more rewarding to work in a ward in which mothers feel at home than the more formal, impersonal type of unit. They should remember, too, that much of their work has good results which they never see—for after the child goes home he will be saved many of the emotional troubles of convalescence if he has never completely lost his parents while in hospital.

A Note on Infant Feeding

This large subject can only be touched on here; nurses are advised to consult one of the many handbooks available for fuller details.

(1) *Breast feeding*.—Breast milk is the ideal food for babies. Its ingredients—protein, fat, sugar and minerals such as calcium and phosphorus—are perfectly balanced for infants; cows' milk has a different composition and so may cause digestive upsets in frail babies. Breast milk is always at the right temperature and is practically sterile. Breast-fed babies are less prone to infections—especially infantile diarrhoea (p. 379)—than bottle-fed infants, probably because bottles and teats are liable to convey infection unless scrupulously sterilised. Successful breast feeding is very much less trouble for the mother, avoiding as it does all the bother with bottles and teats. It is often emotionally satisfying and can give the mother a profound sense of achievement.

Nurses, midwives and health visitors can do a great deal to help mothers to make a success of breast feeding, firstly by their encouraging, optimistic attitude, and secondly by their careful attention to details of technique. *During pregnancy* preparations for breast feeding begin. If the mother has inversion of the nipples (which may make it impossible for the baby to suck) this may be corrected if she wears a plastic nipple shield under her brassière. During the last few weeks of pregnancy it is as well to show the mother how to

express the breast secretion. Regular emptying of the breast at this stage is thought to reduce the likelihood of serious engorgement when lactation begins—a common cause of failure to breast feed. During the lying-in stage nurses can help a great deal by their personal interest in the establishment of lactation, by demonstrating points of technique and by their care in the prevention and treatment of sore nipples and engorged breasts, and, perhaps most of all, by their patience and sympathy with the small difficulties which so upset mothers in the early days after delivery.

Establishment of breast feeding.—If mother and child are both well, the baby should be put to the breast for a minute or two as soon as possible after birth. Thereafter he is suckled four times a day until the milk comes in—generally on the third or fourth day. He should only be allowed to suck each breast for two or three minutes at this stage; prolonged sucking causes sore nipples.

Once the milk has come in he should usually be fed about three hourly (six or seven feeds a day). Many authorities now believe that both mother and child thrive best if the feeding routine is not too rigid. In some institutions, however, regular three or four hourly feeds are recommended.

Both breasts are given at each feed, the baby being put first to the right and left breast at alternate feeds. Ten minutes at each side are usually allowed for very young babies; small, sleepy infants may need longer, but older infants take their feeds very much more quickly. After the first few weeks of life four hourly feeding is usually satisfactory.

Underfeeding.—If the baby screams a great deal, does not sleep for long after each feed, passes small, green motions and fails to gain weight, he is probably underfed. (Overfeeding is very rare among breast-fed babies.) *Vomiting* may also be a sign of underfeeding, as the ravenous child swallows air as he attacks the breast and this blows up the stomach and causes vomiting. If he is being fed four hourly more frequent feeds may be required. A complementary or “topping up” feed, will probably be necessary too. The mother should make up a feed of two or three ounces and let him take as much as he wants after the breast feed. The milk deficiency may not last long and full breast feeding may be resumed later, or complementary feeds may be necessary throughout. Only a small proportion of women who really want to breastfeed their babies prove totally unable to do so.

(2) *Bottle feeding.*—Either a cows' milk mixture or a dried or evaporated milk preparation is commonly given. *Cows' milk mixtures*

are usually satisfactory, except in very dirty homes. Cows' milk has *more protein* and *less sugar* than human milk. To make it more digestible for babies it is *diluted* and *sugar is added*. For the first weeks of life two parts of milk to one part of water are given; later the mixture should be stronger—three or four parts of milk to one of water. By about four months of age undiluted cows' milk can be given. With all these mixtures one and a half level teaspoonfuls of sugar is added to each six ounces of feed. The mixture *must be boiled*, both to sterilise it and to make the curd more digestible.

Dried milks.—These preparations are made by drying and powdering cows' milk and, in some cases, are safer and more convenient than cows' milk itself. *Half cream dried milk* is made from partially skimmed milk and is usually given to babies in the first weeks of life, after which *full cream* milk is given; in some hospitals full cream milk is given almost from the beginning. Dried milk preparations usually contain added vitamins, and, often, added iron.

Dried milk is reconstituted by adding one drachm (a measure) of powder to one ounce of water. (Do not use a teaspoon; teaspoons vary widely in capacity.) Sugar is added as to cows' milk mixtures, unless the particular preparation already contains sugar.

Evaporated milk is diluted by adding two parts of water to one of milk. One to two drachms of sugar per feed are added.

Amount of feed.—A baby needs about $2\frac{1}{2}$ oz. of feed per pound of body weight per day: thus, an eight-pound baby needs about 20 oz. per day or 4 oz. per feed if five feeds a day are given. Some babies, however, need more than this and some need less; so rather more than the calculated amount should be prepared at each feed and the baby allowed to take what he wants.

Vitamin supplements.—From the age of a few weeks bottle-fed babies should be given *one tablespoonful of orange juice* a day (if Government orange juice is used one teaspoonful is given diluted with from four to six parts of water). Babies who are fed on cow's milk mixtures will also need *one teaspoonful of cod-liver oil* daily. (Most dried and evaporated milks contain added vitamin D, so less cod-liver oil will be necessary if the baby is having one of these preparations.) Breast-fed babies do not need these supplements so early, as human milk has a higher vitamin content than cows' milk.

(3) *Mixed feeding.*—Babies eventually need foods other than milk (a) because an all-milk diet will no longer satisfy hunger, (b) because milk (especially cows' milk) contains little iron, and the child who is milk-fed too long becomes anæmic—often profoundly so. Solids may be introduced at any time from, say, three months onwards

—certainly before six months. *Cereals*—groats, “Farex”, semolina and the like—and potatoes provide extra calories and satisfy hunger. Eggs, meat gravy and sieved green vegetables provide iron. Such foods are gradually introduced, one at a time, over a period of weeks. Later, various fruits, milk puddings, steamed fish, bread and butter and other foods are added, until, at a year, the child is having a good varied diet. After the age of nine or ten months breast or bottle feeding can generally be stopped, by which time the child should be able to drink from a cup. Between one and two years the child's diet becomes still more varied, and after the age of two he generally shares in the family meals and can eat almost anything provided for the rest of the family. In addition, he should continue to take about a pint of milk a day in one form or another, as milk is a first-rate source of the calcium so necessary to bone development, as well as of protein and fat. Plenty of milk, or milk dishes, in the diet throughout childhood is to be recommended. Free or cheap milk is available for all infants and children, and this arrangement may well be partly responsible for the marked improvement in child health which has occurred during the past ten or fifteen years.

(1) Rickets

A full-blown case of rickets is now rarely seen in this country, thanks to the better food eaten by our children. Among the underfed children who teemed in nineteenth-century slums it was very common; the German name for rickets—*die englische Krankheit*—(English disease)—is a horrid reflection on our country, now happily undeserved.

Rickets usually affects fat, flabby children between six months and two years of age. Bottle feeding followed by a diet including too much bread, potatoes and porridge, too little milk, butter, eggs, and no cod-liver oil is the usual story. The rickety baby is restless, irritable, sweaty and finicky with his food.

Later it is evident that his *teeth* are slow to appear, the *fontanelle* in the skull is slow to close and various *bones* are deformed. The wrists enlarge and the ends of the ribs form a series of knobs down the front of the chest (“rickety rosary”). If the child can walk he will be bow-legged or knock-kneed. The pelvis flattens, which is serious in girls, as it may later cause obstructed labour. The skull is square-shaped and the back of the skull may feel soft and yielding (craniotabes).

The rickety child has flabby muscles causing potbelly and spinal curvature. Often he is anæmic and his resistance to infection is poor.



Fig. 96. Rickets.

Lower limbs showing gross deformities.
(From Fanconi and Wallgren's *Textbook of Paediatrics*, Heinemann.)

Cause of the Symptoms.—It is well known that rickets is the result of lack of vitamin D (p. 12). This vitamin is essential to healthy bone formation. It helps in the absorption of calcium from the bowel, and also its incorporation into bone. In its absence plenty of bone tissue develops—hence the swollen ends of the bones—but this bone lacks the hardening calcium salts and so is *too soft*—hence the bow legs and other deformities. The poor diet affects the child's general health, so that he is flabby and his disease resistance low.

Treatment.—Large doses of vitamin D are necessary in the form of cod liver or halibut liver oil. The mother must also be advised how to improve the child's diet. *Sunshine* helps to cure rickets, as it enables the skin to manufacture vitamin D. If the legs are bending the child should be temporarily prevented from walking by putting on splints which project beyond the feet. Orthopædic treatment is occasionally necessary.

Prevention of Rickets.—Rickets does not occur in well-fed, well-cared-for children. Infants should be breast fed (breast milk contains more vitamin D than cow's milk) and, when weaned, should be given plenty of milk, butter and eggs, and not too much starchy food.

Vitamin D supplements.—Breast-fed babies do not need vitamin supplements for the first few months of life if their mothers are eating an adequate diet. (Vitamin A and D tablets are available, free, to pregnant women and nursing mothers at Welfare clinics.) Babies who are fed on cow's milk or on any infant food *which does not contain added vitamin D* should be given half to one teaspoonful of cod liver oil daily, or some similar preparation of vitamin D. National dried milk and most proprietary brands of dried milk, however, contain vitamin D, and many cereal preparations are also enriched with vitamins. If the baby is being given one of these foods he may not need any vitamin supplement. A serious disease called infantile hypercalcaemia (excess calcium in the blood) is thought by some to be partly due to the overdoses of vitamin D many of our babies are being given these days.

(2) Infantile Scurvy

Scurvy, a disease caused by lack of vitamin C, was common among the seamen who manned the old sailing ships and lived on a diet of salt pork and ship's biscuit. To-day scurvy, in this country, is practically confined to infants. Under civilised conditions it is almost impossible to avoid eating enough vitamin C to prevent the disease (p. 12). An occasional case of adult scurvy is found among infirm old people living alone or among those on freak diets. Thus,

an immigrant with peptic ulcer, being no linguist, misunderstood his doctor's directions and lived entirely on boiled milk for six months; he developed severe scurvy.

Rickety babies nearly always come from poor homes, but scurvy has been seen in all classes. Thus in a wealthy home the nanny, a stickler for sterility, boiled the baby's orange juice; the baby developed scurvy as the vitamin C had been destroyed by boiling. Like rickets, scurvy is a disease of bottle-fed babies; breast milk contains more vitamin C than cow's milk.



Fig 97 Scurvy.

(Note swelling and discoloration of the gums. (Professor Smellie's case.)

Scurvy starts gradually; the baby becomes irritable and restless, and seems to resent being touched. Finally he screams if merely approached. The *legs* are especially tender and may be swollen. Occasionally there may be some bleeding into the tissues, or blood may appear in the urine. If the child has any teeth the gums may be purple and swollen.

All these symptoms are the result of *hæmorrhage*, due to the weakening of blood vessel walls caused by the vitamin lack. The pain

and tenderness in the legs—often so severe that osteomyelitis is mimicked—is due to bleeding under the *periosteum* (the membrane covering the bone).

Treatment.—Scurvy is rapidly *cured* by large doses of vitamin C. Ascorbic acid (pure vitamin C), 50–100 mgm. daily, is given for a few days. During this time the diet is adjusted; it should include orange, tomato or blackcurrant juice and solids such as baked potato and greens. The ascorbic acid can then be discontinued. Scurvy can be *prevented* by giving orange juice, or some other preparation containing vitamin C, to all bottle fed babies from the first few weeks of life.

(3) Spina Bifida

This is a fairly common congenital disorder, which affects two or three babies in every thousand. A *congenital defect* is one which is present from birth; it is usually the result of faulty development before birth. Congenital defects account for an increasing proportion of the admissions to children's wards. This is because so many infections have been conquered, or at least tamed by chemotherapy.



Fig 98 Spina Bifida, showing a large myelomeningocele in the lumbar region

In spina bifida the development of the *vertebral column* is defective. Certain vertebrae—usually in the neck or near the base of the spine—lack the bony arch which normally protects the spinal cord and its covering membranes (meninges). The unprotected meninges bulge and protrude through the defect, forming a sac which may be several

inches across. The sac contains cerebrospinal fluid (meningocœle), and it may also contain a part of the spinal cord, or nerve roots (myelomeningocœle). A meningocœle may be covered with normal skin and fatty tissue, forming a firm lump, or the skin may be so thinned that the sac looks like a balloon filled with water. In myelomeningocœle there is no skin at all over part of the sac, where nervous tissue is covered only by meninges. Here ulceration is apt to occur, C.S.F. leaks out, and infection is common.

Complications of Spina Bifida

(1) *Paralysis*.—This occurs in most cases of myelomeningocœle, the nervous tissue in the sac being damaged by displacement, injury or infection. In severe cases the legs are totally paralysed and the child is incontinent of urine and fæces. The paralysis is at first flaccid, later, spastic. In less severe cases only the muscles below the knee are paralysed.

(2) *Leakage of C.S.F. and Infection*.—This is common in myelomeningocœle. Often the leak is sealed off by scar tissue and infection remains localised. Meningitis may, however, develop and may be fatal.

(3) *Hydrocephalus*.—This is often associated with spina bifida of the upper cervical region, for the adjoining part of the brain may be abnormal too, obstructing the circulation of C.S.F. Hydrocephalus may be apparent soon after birth, or not until the baby is six or eight months old.

Treatment.—Operation is successful in many cases, especially in meningocœle, where nerve tissue is not involved.

Babies with large myelomeningocœles awaiting operation, or those in whom operation is not advised, often pose a severe problem in nursing care. Ulcerated areas are covered with tulle gras or other suitable dressings, and the whole sac protected with a rubber or plastic dome. Children who develop paralysis will require exercises, physiotherapy, and sometimes orthopædic treatment to help them to walk.

Prognosis.—In the mildest cases the child grows up normally. In the most severe cases, with progressive hydrocephalus, the child dies within a year. In between come those cases who survive to develop severe or mild paralysis. Most of these can eventually be taught to walk and to achieve bladder and bowel control.

(4) Hydrocephalus

This is a fairly common condition, affecting about 1 in 500 infants. The baby usually seems normal at birth, but soon after the head

begins to grow rapidly. The child's eyes turn downward—a very important sign which helps to distinguish hydrocephalus from other causes of a large head. The veins over the scalp are dilated. The anterior fontanelle, which normally closes during later infancy, remains widely open, and the skull bones are separated by wide sutures.



Fig 99 Hydrocephalus

In about 40 per cent. of cases the head stops growing eventually, the eyes straighten and the child grows up normally, or slightly backward mentally. In more severe cases there is spastic paralysis of the lower limbs. In the most severe cases the skull continues to grow, and blindness, mental defect and severe paralysis develop. The majority of such patients die in early childhood.

Cause of Hydrocephalus.—This is nearly always a *blockage in the circulation of the cerebrospinal fluid*. The C.S.F. is formed in special tufts of blood vessels called the *choroid plexuses*, which project into

the ventricles of the brain. It flows from the ventricles to the space between the arachnoid membrane and the brain and spinal cord (p. 291). It is eventually re-absorbed by the capillaries in the arachnoid covering the brain. If adhesions form in any part of its course it may be dammed back. The ventricles dilate, the surrounding brain tissue expands and thins out. As the bones of the skull have not yet fused in the infant, these bones are pushed apart and the head enlarges. The thinning of the brain tissue may be severe enough to cause mental defect and paralysis.

The adhesions causing hydrocephalus are usually caused by low-grade meningeal infection, which may occur at or soon after birth without any sign of meningitis. Frank meningitis in infancy will also cause hydrocephalus (p. 324). Hydrocephalus may also be caused by a congenital abnormality of the meninges or ventricular system. It is often associated with spina bifida (see above).

Treatment.— In many cases hospital treatment is unnecessary: if the child is mentally backward or has spastic legs special training and education may be required. In *progressive hydrocephalus* operation may be necessary to establish a new channel through which the dammed-up C.S.F. may drain. Many procedures have been tried and the results of some of the newer operations seem promising.

(5) Cerebral Palsy

This is a fairly common disorder; there is about one child with cerebral palsy in every 1000 children of school age. There are two main varieties—spastic paralysis (Little's disease) and athetosis.

Spastic Paralysis

Symptoms.— Usually the mother first notices that her baby is *backward*; he is slow to sit up and slower still in learning to walk; often he is slow to talk. The child may not walk till he is five or later. It soon becomes apparent that the child's muscles are *weak*, and usually *stiff*. If the baby is held by the chest and lifted up, the head droops, the arms are held close to the body and the legs are straight and pressed closely together. In older children the stiffness becomes more obvious; the thighs are often pressed together so tightly that when the child learns to walk he crosses one foot over the other with each step (scissor gait). The legs are almost always affected more severely than the arms, which often escape altogether.

If the legs only, or legs and trunk, are affected, the condition is called a *paraplegia*. In *tetraplegia* all four limbs are affected. *Hemiplegia* may also occur, paralysis affecting one side of the body only.

Hemiplegia may be congenital, or may develop suddenly during infancy or early childhood—often after a series of convulsions.

Many spastics are mentally defective—about 50 per cent. compared with 3 per cent. of the “normal” population. As a rule, the worse the paralysis the worse the mental defect. Many spastics are epileptic too.

Athetosis

Here, too, the child is backward—in learning to sit up, to walk and to talk. He also shows *abnormal movements*. At rest, his head jerks, and his hands and arms twitch, jerk or writhe. As soon as he tries to do anything the movements get worse, and these uncontrollable movements are often as disabling as the stiffness and weakness of spastic paralysis. The face and tongue are often affected; the child grimaces and slobbers and normal speech is impossible. The movements cease during sleep.



Fig. 100. Athetosis
Hand during athetoid movements.
(From Fanconi and Wallgren's *Textbook of Paediatrics*
Heinemann.)

In athetosis the neck and trunk muscles are usually weak. The limbs may be rigid or limp, or the tension may vary from time to time.

The athetoid is often partially or severely *deaf*.

These children may be mentally defective, but a high intelligence may be masked by crippling athetosis. A child who grimaces and dribbles, who cannot feed himself or speak intelligibly at six may look an imbecile, though special tests, making due allowances for handicaps—including deafness, if present—show that he is highly intelligent.

The Cause of Cerebral Palsy

This is an *abnormality of the brain*—sometimes confined to the parts of the brain controlling the movement of muscles, sometimes widespread, causing deafness, mental defect and other symptoms. The brain may *develop abnormally* before birth, or it may be *damaged at or soon after birth*. The damage may be the result of a difficult forceps delivery, or of asphyxia, or of neonatal jaundice and kernicterus (p. 383). Athetosis is nearly always the result of such brain damage. In spastic paralysis there is often a history of prematurity or abnormal labour, but in many cases there is nothing to suggest brain injury, and abnormal brain development before birth seems to be the cause.



Fig. 101. Pyloric Stenosis.
Note visible peristalsis (Professor Smellie's case)

Treatment.—About half of cerebral palsied children need prolonged special treatment—the others are the mild cases, and at the other extreme those whose intelligence is too low for them to respond to treatment. The treatment of a child with cerebral palsy involves the parents, doctors, and physiotherapists; often, speech therapist and psychologist; and, for children of school age, the child's teacher. These children often improve enormously with the help of equipment which is adapted to their special needs (chair, mug, spoon,

etc.) and of physiotherapy and exercises which help to develop normal posture and movements. They need constant encouragement, without too much pushing and stress—athetoids are always worse when strained and nervous.

The more severe cases should be educated in special schools. Physiotherapy, speech therapy, etc. may be given at school or in hospital departments.

Orthopædic treatment is sometimes necessary; the child may require a caliper or support, or an operation may be advisable.

Prognosis.—Mild cases can often attend normal schools and live a normal life. Moderately severe cases may be fitted for normal life, and for earning a living, after prolonged treatment and special education. The most severe cases will always have to be looked after by relations or in institutions. A proportion of these very severe cases die young.

(6) Congenital Hypertrophic Pyloric Stenosis

This remarkable disease affects young infants; curiously enough the patient is generally a boy, most often his mother's first baby. She will tell us that he did well for two or three weeks, perhaps longer, when suddenly he began to vomit—one feed was taken normally and the next violently returned. Since then he has vomited every feed; scarcely has he finished before the whole feed is "pumped back," after which he is ravenous for more. His bowels are constipated and, if he has been long untreated, he will have lost a lot of weight.

The cause of the trouble is an obstruction at the outlet of the stomach (the *pylorus*). The ring of muscle surrounding the pylorus is enormously thickened (*hypertrophic* means overgrown) so that food cannot get out of the stomach and is returned. The mass of tissue can be felt as a little lump just below the right ribs. If the baby is examined just after a feed waves of peristalsis can be seen passing from left to right across the upper abdomen. These waves represent the powerful contractions of the stomach as it tries to force its contents past the obstruction. The cause of the pyloric overgrowth is quite unknown.

Treatment.—The majority of cases are treated surgically, the thickened ring of muscle being slit at operation. *Rammstedt's operation*, as it is called, has a mortality of less than 1 per cent. in hospitals where pædiatrician, surgeon and nursing staff work together as an experienced team. The baby need only be in hospital for three days—the longer he is in the ward the more likely he is to pick up some infection. If he is breast-fed—which he should be—the mother

should be admitted too. Before the operation an intravenous drip may be necessary, if excessive vomiting has made the baby dehydrated. Just before the patient goes to the theatre his stomach is washed out. Four hours after the operation the baby gets an ounce of sterile water. He is fed two-hourly for the next two days starting with one ounce of half-strength expressed breast milk (or similar feed) and working up to two-ounce full-strength feeds. On the third day—or sooner in some hospitals—he is put back to the breast, or given a full-strength bottle feed, the proper amount for his age and weight being given in seven three-hourly feeds; if all is well he is then discharged, returning on the eighth day to have the stitches out.

Medical treatment.—This is sometimes recommended for older, more robust infants in whom the obstruction is not severe. Atropine methonitrate (“eumydrin”) or hyoscine methonitrate (“skopyl”) is given; these are anti-spasmodics and help to relax the pyloric muscle and so relieve the obstruction. Stomach washouts and graded feeds will also be necessary.

Fortunately, whichever treatment is used, those babies who recover do so completely.

(7) Cœliac Disease

This is a disease in which the child fails to absorb certain food substances. Symptoms appear soon after the baby begins to take a mixed diet. He loses his appetite, seems off colour, and develops *diarrhœa*. The stools are large, pale and offensive. The child stops growing and gets thinner and weaker; the wasting of the buttocks is particularly striking and contrasts with the distended abdomen. Usually he becomes irritable, difficult, hypochondriac and unnaturally precocious. A child of three, on admission to hospital, summoned the sister and asked the house physician's name, for, said she, she wished to know whom to send for should she be taken ill in the night.

Untreated cœliac disease lasts for years, often into adolescence or adult life. Rickets and respiratory infections used to be common among these children. Fortunately the outlook is now very much brighter.

Cause of the symptoms.—The diarrhœa and pale, offensive stools are due to the child's failure to absorb the fat in his food (the stools may contain twice as much fat as normal). Carbohydrate absorption is also upset; the child fails to thrive as he does not absorb sufficient nourishment. It has recently been shown that this digestive disturbance is caused by *gluten*—a protein present in wheat and rye flour.

Exclude gluten from the child's diet and he thrives; allow gluten-containing foods in any form and he relapses. How gluten brings about this disturbance is not yet known.

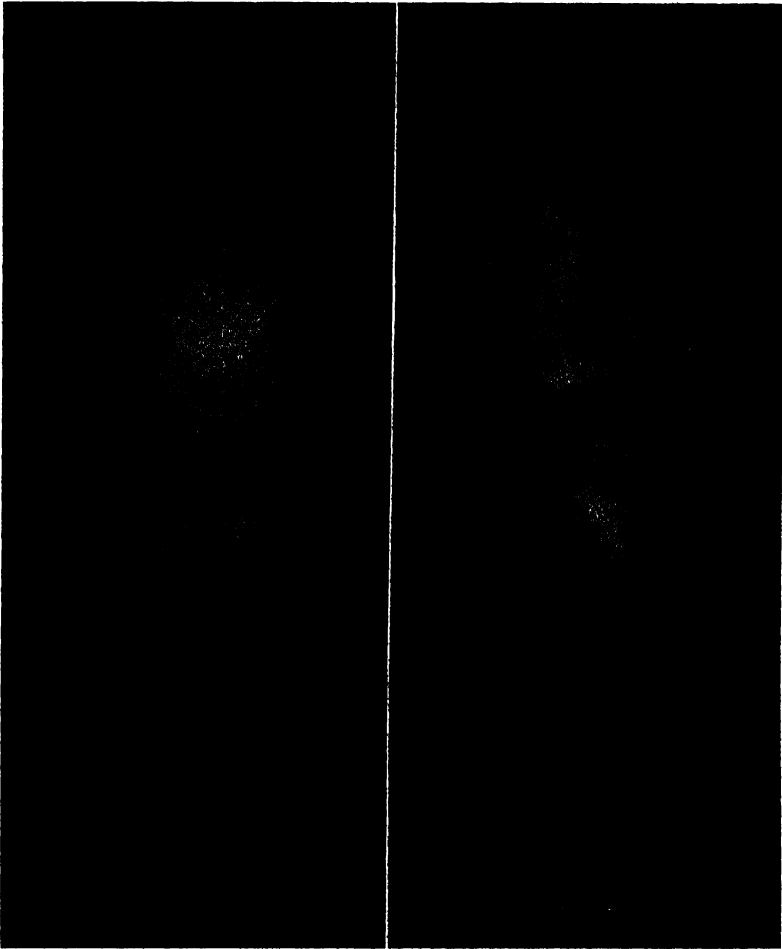


Fig. 102 Celiac Disease.

Note wasted buttocks and protruding abdomen. (Professor Smellie's case.)

Treatment.—A diet excluding *wheat flour* (and rye flour) is necessary. The child may eat all other foods; fat-free diets are no longer recommended.

Ordinary bread, biscuits, cakes, pastry, macaroni and semolina are forbidden. Gluten can be removed from wheat flour, and the resulting "wheat starch" can be used for making loaves, biscuits and

puddings. Cornflour, oatmeal, and ground rice can also be used for cooking, but custard powder and ice-cream contain wheat flour and are forbidden. The patient must keep to this diet throughout

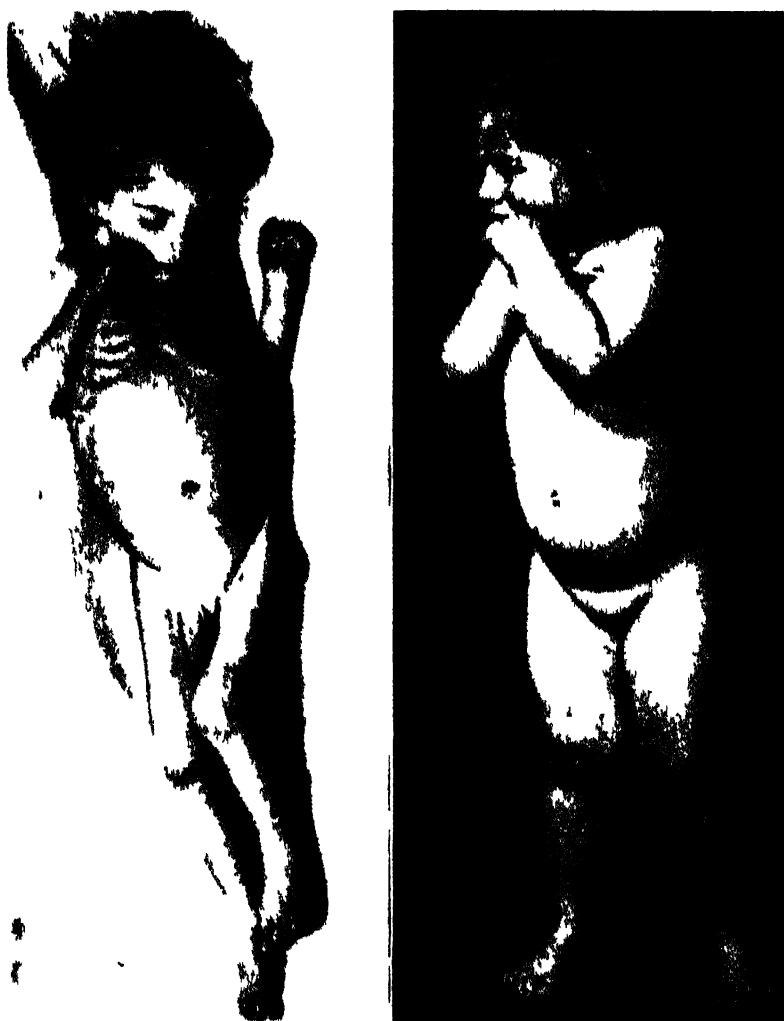


Fig 103 Celiac Disease
Child before and after treatment with gluten-free diet (Professor Smellie's case)
(Reproduced by permission of the editor of *The Lancet*)

childhood, perhaps even into adult life. The prognosis is then good, and normal health and development are to be expected.

(8) Fibrocystic Disease of the Pancreas

This disease tends to run in families, and two or more brothers or sisters or cousins may be affected. The baby usually seems normal at birth, but soon after begins to have loose, pale stools; although his appetite is good he fails to gain weight or may lose weight. Finally he may be severely wasted. Repeated *respiratory infections* also occur, and these may disturb the child's growth and development even more than the digestive disorder. Those who survive infancy usually develop bronchiectasis.

In other cases symptoms develop within a few hours or days of birth. Nothing is passed per rectum and the abdomen rapidly becomes distended; hard lumps can be felt in the distended abdomen. The baby may die of acute intestinal obstruction. This condition is called *meconium ileus*.*

Diagnostic Tests.—If a specimen of *duodenal juice* is examined it is found to lack *trypsin*—the pancreatic enzyme which digests protein. This is the most certain test for fibrocystic disease, but as the withdrawal of duodenal juice is difficult for the doctor and uncomfortable for the child, simpler tests are used when possible.

Fæces.—These contain excess fat. They also contain *undigested meat fibres* which may be seen under the microscope. More important, the fæces, like the duodenal juice *lack trypsin*; solutions of the fæces do not digest protein such as gelatine.

Sweat.—This contains *more salt* than normal sweat. The child's "finger prints" are taken on a plate of jelly containing silver nitrate; the excess salt reacts with the silver nitrate to make the finger prints white. This is a particularly useful test as it can be done in a moment in ward or out-patient clinic.

Explanation of the Symptoms and Signs

The child wastes because his pancreatic juices do not digest his food normally; the lack of trypsin is shown in the tests described above, but other enzymes are affected too. Post-mortem examination shows that the pancreatic ducts are obstructed by abnormally thick, sticky mucus, which may collect in cyst-like spaces, surrounded by fibrosis (hence the name fibrocystic disease). Cells producing mucus are affected in other parts of the body, particularly the bronchi; small bronchi become obstructed and collapse, and bronchiectasis follows. Abnormally tough mucus may block the bowel of the newborn, causing *meconium ileus*.

*Meconium: A blackish substance present in the intestines at birth. Ileus: Obstruction of the bowel.

The exact cause of the sweat abnormality is unknown.

Treatment.—This aims at *maintenance of nutrition* and *control of respiratory infection*.

Nutrition.—The diet should be ample, and plenty of protein foods must be included. If the stools are very fatty (steatorrhœa) the dietary fat may be reduced, but not drastically. *Pancreatin* is given in the form of granules or tablets; this helps to replace the missing pancreatic digestive enzymes.

Infection.—Even mild respiratory infection requires intensive antibiotic treatment. Throat swabs may identify the responsible organisms, and this helps to determine which antibiotic should be given. Small daily prophylactic doses of antibiotics are sometimes recommended.

Treatment of Meconium Ileus.—Surgery may be required to relieve the obstruction.

Prognosis.—These children used to die in infancy or early childhood. Modern treatment usually prolongs life and health for many years, though even now their lives are precarious as they grow older. It is too early as yet to forecast the future for the children now being treated for fibrocystic disease.

(9) Pink Disease

Although the exact cause of pink disease is unknown there is good evidence that it is due to *mercury poisoning*. Calomel (mercurous chloride) used to be the main ingredient in so-called “teething powders”, and it was found that many babies with pink disease had been given these powders regularly and in large doses. Fortunately, only a very small proportion of all babies receiving mercury-containing powders develop pink disease. The disease has recently become extremely rare.

The onset of the disease is generally between the ages of 9 months and 2 years. Symptoms come on gradually, often after some vague feverish illness. The child becomes increasingly *miserable, weak* and *restless*. No one is more wretched than a child with pink disease. He hates the light and burrows into the pillow, whimpering perpetually, especially if disturbed. The *skin* is itchy, especially that of the hands and feet, which are swollen and reddish pink—hence the name of the disease. In his distress he rubs, scratches and even bites the itchy parts and may tear out his hair in handfuls. His *mouth* is inflamed and the *teeth* may fall out and be swallowed. The *muscles* are flabby and toneless. The *pulse rate* is invariably raised and is usually 140 or more.

These children have little resistance to infection; skin sepsis is

common and they may succumb to pneumonia. Unless such infection proves fatal the disease invariably clears up completely in three to nine months.

Treatment.—No curative treatment is known. The patient should not be admitted to hospital, because of the risk of infection, but a nurse to help the distracted mother is desirable. Silk or cotton clothing reduces the skin irritation, which may also be allayed by tepid baths followed by a methylated spirit rub and powdering with talc powder; calamine lotion is soothing too. A sedative such as chloral or phenobarbitone is generally necessary.



Fig. 104. Pink Disease (Professor Smellie's case)

The child has no appetite, and his food must be prepared and offered with skill and imagination. Ice-cream, jellies and sandwiches may prove acceptable when homelier fare is rejected, and a very good diet can be given in this way. Vitamin supplements are essential; large doses of vitamin B₁ by injection have been recommended.

Prevention of pink disease.—Calomel should not be given to young children. (Reputable firms manufacturing "teething powders" now omit calomel—this may account for the present rarity of pink disease.) Young children do not need regular aperients and this is all most "teething powders" are. Nurses should try to dissuade mothers from dosing their children with doubtful remedies. They are mostly useless and a few may be harmful. The danger of calomel has only recently been recognised; it is quite possible that other popular medicants may prove harmful too. As already noted (p. 365) even vitamin D can do harm in over-generous doses.

(10) Infantile Diarrhœa (Gastro-enteritis)

Infants may have loose, frequent motions because they are unsuitably fed; diarrhœa may accompany almost any type of infection,

such as otitis media, or may be caused by dysentery organisms, as in adults (p. 321). Finally, there is a variety of diarrhœa peculiar to infants. It is highly infectious, highly fatal, is almost confined to bottle-fed babies and is prone to attack the infants in institutions such as infants' wards, day nurseries, or the nurseries in maternity units.

The cause of this dreaded infection is probably a special variety of *Escherichia coli* (formerly called *B. Coli*), a near relation of the organism which lives harmlessly in healthy adult bowels.

A baby with gastro-enteritis ceases to thrive, loses weight, vomits his feeds and has frequent loose, green, offensive stools. In severe cases the vomiting and diarrhœa get worse and the baby goes rapidly downhill. He lies restlessly in his cot; his face is pale but his dry lips are unnaturally red. His brow is furrowed, his expression anxious, and he wearily turns his sunken eyes from side to side as though in search of comfort. The fontanelle is sunken and the skin inelastic, so that, if you pick up a fold of skin it does not immediately spring back when released. These are signs of *dehydration*, the tissues being depleted of water by the constant sickness and diarrhœa. The stools may make the buttocks sore. In the last stages orange stools and blood-stained or "coffee ground" vomiting may herald death.



Fig. 105. Infantile Diarrhœa showing severe wasting and dehydration (Professor Smellie's case)

Treatment.—Good nursing is all-important, and doctors and nurses must work as a team. In *mild cases* the baby may do well if given clear fluid to drink instead of milk for 24–48 hours. The fluid may be 5 per cent. glucose or table sugar (1 tablespoonful to the pint) plus 0.3 per cent. salt (one-third teaspoonful to the pint), or even plain water. Diluted milk is then given and the strength of the feed is gradually increased. In *severe cases*, where the baby is vomiting much, he cannot be fed by mouth and the fluid he needs so badly must be given by *intravenous drip*. He can do without food for a few

days but he will die without fluid. An intravenous drip is given by means of a needle inserted into a vein in the ankle or arm or scalp.

The fluid given generally contains glucose (5 per cent.) plus various sodium and potassium salts, to replace the salts lost in the vomit and stools. The exact composition depends on the degree of dehydration and the length of time the drip has been running.

The *rate of flow* of the fluid depends on the size and illness of the baby; it should be carefully watched, for *too much fluid* would drown the baby and *too little* will not relieve dehydration. The fluid is run in fairly fast at first, to relieve the dehydration, then more slowly. For a severely dehydrated baby weighing 10 lb. about 100–200 ml. should be given in two to four hours (10–20 drops per min.) and the rate then slowed.

An intravenous drip can be kept running for days if necessary; the site of the drip must be scrupulously protected against infection (see p. 43). When the baby has stopped vomiting, and when his dehydration has been overcome, as shown by his general condition and the elasticity of his skin, small feeds by mouth may be cautiously tried, starting with a very dilute feed, and working up gradually to the proper amount for his age and weight.

Sore buttocks may be treated with zinc and castor oil, or gentian violet; exposure of the buttocks for part of the day is helpful. Infections of all kinds are common in infantile diarrhoea; if present the appropriate treatment must be given.

Drugs.—Sulphonamides, tetracyclines and neomycin have all been used with varying success in different epidemics.

Prevention of Infantile Diarrhoea. Babies who are *breast fed* and who are *not in an institution* do not get gastro-enteritis. So babies should not be admitted to an institution unless absolutely necessary; if admitted they should be discharged as soon as possible. The prevention of infective diarrhoea in an infants' ward or nursery calls for the most scrupulous barrier nursing (p. 42). Unfortunately, although the spread of *Sonne* dysentery and many other infectious diseases can be prevented thus, *E. coli* enteritis seems to spread in spite of these precautions. Rectal swabs taken from all babies on admission, and the isolation of all carriers of recognised disease-producing strains and all babies with diarrhoea may keep the infection from entering the infant's ward.

(11) Intussusception

This is a form of acute intestinal obstruction which is commonest in babies aged about five to seven months. The obstruction is caused

by a portion of bowel which enters the bowel immediately beyond—is *invaginated* into it. Usually the terminal portion of the small intestine is invaginated into the large bowel. The cause of intussusception is unknown. A digestive upset may precipitate it: an intussusception usually occurs at the age when babies are beginning to take a more varied diet.

The history is often very characteristic. A previously healthy baby suddenly has attacks during which he screams with pain and draws up his legs. In between attacks he seems perfectly well. He may be *constipated*, or he may pass one or two normal motions in the early stages, after which there will be no further bowel action. Sometimes a little *blood and mucus* are passed from the rectum.

As the hours pass the attacks of pain become more and more frequent; the baby begins to vomit and at length becomes shocked and dehydrated.

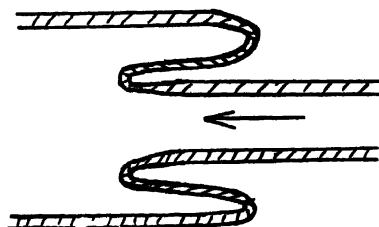


Fig 106 Diagram of an Intussusception

The diagnosis is confirmed if a *sausage shaped tumour* can be felt in the abdomen, representing the intussusception. Or the invaginated bowel may advance so far that it can be felt on *rectal examination*, or the tip of the examining finger may be bloodstained.

The treatment is surgical. If the baby is dehydrated or shocked a pre-operative intravenous drip is given. If the operation is begun within twelve hours of the onset of symptoms it is usually quite easy to withdraw the invaginated bowel and restore it to its normal position. After such an operation the child recovers rapidly and the outlook is excellent. But if operation is delayed the affected bowel may become gangrenous, owing to the gradual cutting off of its blood supply. At operation the gangrenous portion will have to be resected—a very grave undertaking—and the baby's chances of recovery are much less.

Occasionally intussusception recurs, but a second operation quickly corrects it.

Sometimes in early cases, a barium enema will correct an intussusception, and no operation is necessary.

(12) *Hæmolytic Disease of the Newborn*

A mother has one or two normal babies. Then she has a baby who is severely anæmic, or jaundiced, or œdematous, or stillborn. She may have a succession of such babies; often each baby is more severely ill than the last, though this is not always so.

That is the characteristic history of a family affected by hæmolytic disease of the newborn.

The usual cause of this disease is a difference in blood group between mother and child. For instance, if the father's blood is Rh positive and the mother's is Rh negative, the child may inherit the father's group. During pregnancy, should some of the baby's Rh positive red cells leak through into the mother's circulation, *anti-bodies* to the baby's red cells may develop in the mother's blood (just as antibodies to the invading organisms are produced during an infection). Should these antibodies traverse the placenta and enter the baby's blood stream they destroy his blood cells. This causes anæmia, and, if the pigments from the broken-down red cells cannot be quickly excreted by the liver, jaundice develops.

In the severest cases the baby is born dead with severe anæmia and œdema- hydrops foetalis.

The baby is apt to be more severely affected in later pregnancies because more and more antibodies are produced with each pregnancy. The first baby is rarely affected.

The symptoms are as follows.

(a) *Hæmolytic Anæmia*. The baby may be pale at birth, or rapidly becomes so. The general condition is good.

(b) *Icterus Gravis Neonatorum*. (Severe jaundice of the newborn.) The baby seems normal at birth, but within a few hours, or less, he becomes deeply jaundiced. Jaundice may persist for weeks, followed by gradual, complete recovery. But in some cases the baby, when about three or four days old, suddenly deteriorates. He goes off his feeds, throws his head back, goes blue or has a convulsion. This condition is called *kernicterus**; most of these babies die; some apparently recover. Many of these later show signs of *athetosis* (p. 371), sometimes with mental defect and/or deafness.

Treatment of *Hæmolytic Disease*

(a) *Treatment of the baby*. If the baby is anæmic or jaundiced an *exchange transfusion* is carried out. This means that the

* *Kernicterus*: Kern=nucleus; icterus=jaundice. In babies dying of kern-icterus certain parts of the brain are found to be bile-stained—hence the name.

baby's Rh positive blood is exchanged for Rh negative blood, which will not be harmed by the antibodies the child received from his mother. Whenever hæmolytic disease is suspected, the cord blood is tested for hæmoglobin and bile pigments—the results show whether exchange transfusion is necessary or not. When necessary, exchange transfusion is carried out as soon as possible after birth. Plastic tubing is introduced into the umbilical vein and the baby's Rh positive blood withdrawn with a syringe and replaced with Rh negative blood.

Only a minority of affected babies need an exchange transfusion. Some of the remainder may need a straight transfusion later on.

(b) *Treatment of the mother.* If a woman is known to have Rh negative blood and, during pregnancy, Rh antibodies are found in her blood, she should be delivered in a hospital where an exchange transfusion can be carried out if necessary. Unfortunately, no method is yet known of preventing hæmolytic disease of the newborn.

The importance of Rh grouping of blood before transfusions has already been mentioned (p. 160). Should a transfusion of Rh positive blood be given to a woman whose blood is Rh negative her chances of bearing a child with hæmolytic disease are seriously increased.

Prognosis

The Outlook for the Baby. Babies who are *œdematous* at birth nearly always die within a few hours. Babies who are *anæmic only* nearly always recover. In between come the jaundiced group; *with adequate exchange transfusion the great majority recover completely.* But some of these babies die, and a few of the survivors develop athetosis (p. 371)—especially if treatment is delayed or inadequate.

The Outlook for the Mother. A mother who has had an affected baby is likely to have the same trouble with future babies. It is most important for nurses to realise, however, that in about ten per cent. of all married couples the wife is Rh negative and the husband Rh positive, and that *the vast majority of these couples will never have a baby with hæmolytic disease.* Many women who know that they are Rh negative have exaggerated fears, and may actually believe that they are unable to bear normal children; it is most satisfactory to be able to dispel these gloomy illusions.

THE SOCIAL ASPECTS OF DISEASE

AN illness seldom has a single cause. Pulmonary tuberculosis is caused by the tubercle bacillus; but tubercle bacilli enter the lungs of thousands of people who never develop the disease. A poor person is more likely to develop pulmonary tuberculosis than a rich one; there is a special risk to those who live in the same house as a tuberculous patient; men whose work exposes them to silica dust—such as quarry men and miners—are also likely to develop the disease. The study of such factors is often known as *social medicine*. In studying social medicine we consider how heredity, family life, work, housing, leisure activities and the like affect a person's susceptibility or resistance to disease.

Social factors in illness have often been mentioned in this book. Rheumatic fever (p. 57), pneumonia (p. 94), bronchiectasis (p. 104) and pulmonary tuberculosis (p. 108) are all commoner among poorer folk. This may be because such people are likely to be undernourished, with a poor resistance to infection, or because they live in overcrowded houses, with many chances of passing on infections. Probably both food and housing are concerned. Iron deficiency anæmia (p. 142) is certainly attributable to a poor diet, as is rickets (p. 363). All these diseases are becoming less common or less severe as social conditions improve.

On the other hand, poliomyelitis (p. 291) flourishes among the well fed, well housed populations of civilised countries. Hypertension (p. 65), coronary artery disease (p. 71) and duodenal ulcer (p. 164) are all "diseases of civilisation"; all have recently become commoner, and are either equally common among all classes or, like coronary occlusion (p. 71) they affect chiefly those in well paid, responsible posts. Another disease which has recently become alarmingly common is cancer of the lung (p. 122); this is largely due to the great increase in cigarette smoking over the past few decades.

In many illnesses the *family history* is important. Certain illnesses, such as migraine (p. 313), asthma (p. 134), urticaria (p. 268), eczema (p. 270), diabetes (p. 237) and epilepsy (p. 310) may run in families. Pulmonary tuberculosis (p. 108) may be passed on from one member of the family to another; the disease is not inherited, but there may be less resistance to the disease in some families and races than in

others. Many acute infections, of course, affect several members of the family in quick succession. Sometimes the same infection may have different effects in different members of the same household—for instance, hæmolytic streptococci may cause scarlet fever, tonsillitis, otitis media, erysipelas, or whitlow (see p. 355).

The family history is particularly important in cases of mental ill health (p. 389). Certain mental characteristics may be inherited; also, people are profoundly affected by their upbringing and family life, which largely depend on the parents' psychological make up. For both these reasons neurotic parents often have neurotic children.

These examples show that it is often impossible to study a single case of disease in isolation. Before we can answer the question "Why has this person developed this particular disease?" we may have to inquire into his family history, his work, his housing, his milk supply (p. 121), his water supply (p. 316) or whether he has ever had any protective inoculations (pp. 175, 294, 320).

Public Services which promote health

It is difficult to realise that, until about a hundred years ago, the community took practically no responsibility for the health of its members. Today we believe that the existence of preventible illness is a slur on the community—"if preventible, why not prevented?"—and that treatment should be available to all who need it. Here are a few of the ways in which the health of the public can be improved or maintained, and illness and distress relieved.

Improvements in housing and sanitation have probably saved more lives than the most spectacular medical discoveries. In this country cholera has been entirely and typhoid (p. 316) very largely wiped out by improved sanitation. Improved housing also reduces the incidence of respiratory infections such as tuberculosis. Local authorities subsidise the building of new houses for people who need them; they also appoint sanitary inspectors to supervise housing and sanitation.

The *health of the individual* is promoted by the various local authority welfare clinics and their officers. At infant welfare clinics mothers are given advice on child care; ante-natal clinics care for pregnant women; the school medical service looks after the health of school children. Inoculations against diphtheria and whooping cough and vaccination against smallpox are available for all, and mothers are urged to have their children protected against these diseases. B.C.G. (p. 120) is available for all tuberculosis contacts and other groups, such as nurses, at special risk. All school children are

offered B.C.G. at the age of thirteen if they are tuberculin negative. Poliomyelitis vaccine is available for the under-forties (after forty the infection is uncommon). *Health visitors* play a most important part in all these services, especially in teaching mothers how to care for their children's health.

For those who are ill the National Health Service provides all necessary treatment. To student nurses the hospitals are, of course, the most familiar part of the service. But it must be remembered that a far greater number of sick people are treated at home by general practitioners. Here the district nurses play an invaluable part. At present there is too little co-operation between the health services inside and outside the hospital. Whenever such co-operation is increased the patient benefits. In some hospitals health visitors attend rounds in the children's wards, and meet the patients they will later see in the course of their home visits. Co-operation between midwife and health visitor is especially important, whether the baby is born at home or in hospital, if there is to be proper continuity of care for the mother and child.

Hospital nurses have a responsibility to their patients which does not end with the nursing of one particular illness. Patients often find it easier to talk to nurses than to doctors, and nurses who are aware of their patients' problems outside the hospital can often give them valuable advice about what they should do on discharge, and on health problems generally. It is most helpful if they can tell their patients what facilities for treatment and after-care are available after they leave the hospital. Often such information is chiefly the concern of the almoner, but nurses see more of the patient than does any other member of the team, so that it may be easier for them to impress on him the importance, say, of attending the follow up clinic or of calling in his private doctor after he leaves hospital.

The patient usually comes under the care of his family doctor on discharge from hospital. If nursing treatment is still necessary this is given by the district nurse. He may also require out-patient treatment at hospital—for instance in the physiotherapy department. As already described, special arrangements are made for tuberculous patients, whose treatment is supervised by the physicians attached to the chest clinic. The tuberculosis nurse or health visitor is an important member of the chest clinic team.

People who are ill or disabled are also helped by the State by means of allowances and pensions. Wage earners who are ill receive sickness benefit; the war disabled receive pensions; those whose work caused their illness or injury receive workmen's compensation.

Financial help is also given to the parents of more than one child (family allowance), to the unemployed (unemployment benefit) and to those who have retired (old age pensions). National assistance is available for those in distress, when other allowances are unavailable or inadequate.

These subjects are of importance to nurses, partly because they, as citizens, both use and help to provide these services, but chiefly because whatever affects their patients' health is their concern. An understanding of the social aspects of disease thus helps them to help their patients, and so makes them better nurses.

CHAPTER TWENTY-FOUR

MENTAL ILL-HEALTH

BY PORTIA HOLMAN, M.D., F.R.C.P.

Introduction

A TEXT-BOOK such as this is no place for a full account of psychological medicine or of the science of psychology. All that will be attempted is a brief discussion of the nature and causation of mental ill-health and of the methods commonly used in treating it.

WHAT IS MENTAL ILLNESS?

It has often been pointed out that some of the conditions that seem to us to be insanity or serious mental illness would not be so regarded in other countries or even, at other periods of its history, in our own.

Much mental illness is then, behaviour that seems strange in the society in which it occurs; but some mental illness is a consequence of the way of life in the country in which it occurs (e.g. when we say a breakdown is "due to the strain of modern life", this is what is meant). Many thoughtful people have criticised the organisation of our society, saying that some of its demands are unreasonable or unnecessary and that, if civilised life were less exacting, mental health would be improved.

Be that as it may, most people are able to accept most of society's demands most of the time, and are able to react to them *appropriately*. By *react* we mean to think, feel and behave; by *appropriately* we mean suitably in the circumstances; e.g. appropriate behaviour is of one sort at a funeral, of quite another sort at a party, and of still another in the ordinary working day.

CAUSES OF MENTAL ILLNESS

Since everyone makes inappropriate reactions sometimes, to do so does not, in itself, denote any departure from normal mental health. It is only when inappropriate reactions occur more often than appropriate reactions, or when they occur systematically in certain circumstances, that they may be regarded as evidence of mental illness. There is no absolutely hard and fast line between mental health and ill-health; but, while at the border-line there is room for differences

of opinion, the further the patient moves from the border, the easier it is to reach a decision about him and the more certain it is that different observers will agree.

Those who react inappropriately more often than not may be termed *psychotic* (suffering from psychosis, Gr. an illness of the *psyche*, the mind), those whose inappropriate reactions are less frequent but systematic may be termed *neurotic* (suffering from *neurosis*, an illness of the "nerves"—not of the anatomical nerves, but the nerves in the figurative sense).

Opinions differ about the causes of psychoses and neuroses, but most people would agree that the psychoses, at least, are of two very different types with different types of causation: those where there is a recognizable bodily change (usually in the brain or anatomical nervous system) and those where, by the means available to us at present, no bodily changes can be detected. For convenience, we may call these two types (1) organic, and (2) psychogenic (of psychological origin). It must be remembered that, although in the second group no organic cause has been detected, this does not mean that there is none.

We can therefore classify the causes of mental illness as:

I. ORGANIC.

II. PSYCHOLOGICAL.

On the organic side the principal causes are:

- (1) Loss of brain tissue.
- (2) Damage to brain tissue by toxins.
- (3) Interference with brain tissue by changes in blood chemistry.
- (4) Endocrine disturbances.

On the mental side we distinguish:

- (1) Inborn make-up.
- (2) Environmental factors.
- (3) Psychological conflict.

IT MUST ALWAYS BE REMEMBERED THAT ANY OR ALL OF THE FORE-GOING CAN BE ACTING TOGETHER.

ORGANIC MENTAL ILLNESSES AND PSYCHOSES

The brain is the organ concerned with mental processes. It is not, then, surprising that when the brain is diseased, there will be

disturbance of mental function as well. Our knowledge of how functions are localised in the brain has been gained by a study of patients in whom some part of the brain has been affected by injury, tumour, vascular disease, etc. In this way we have learnt that there is a special part of the brain in control of motor functions, another where sense impressions are recorded, that there is a small area known as the speech centre and that the general adaptation of the person to the world around him depends largely on his frontal lobes.

GENERALISED BRAIN DISEASE.—Diseases such as arterio-sclerosis syphilis, encephalitis and meningitis affect the brain as a whole and cause severe mental changes.

LOCAL INJURIES.—After a head injury, a patient may show various losses of mental power according to which part of the brain is affected. Children injured at birth may go through life with certain sorts of specialised defects (see p. 370). The curious disabilities known as word blindness and word deafness are due in all probability to injuries at this time. A tumour or any other disease that picks out certain parts of the brain will result in similar losses of special functions.

ORGANIC PSYCHOSES.—If mental changes are severe enough, the patient is said to be psychotic. In the psychoses thinking, feeling and behaviour are all, to some degree, inappropriate. This is true of the brain diseases just mentioned and is clearly seen in the senile (Latin, *senilis* = aged) psychoses of old people. In many old people there is generalised brain degeneration: they tend to suffer from loss of memory, confusion about their whereabouts in space, confusion about what time, day, month or year it is, and about whether something that happened was yesterday, last week or ten years ago. They are then said to be *disorientated* in space and time. They may not be able to find their way about or remember where they live. They may reach a state when they cannot say who they are, or give any account of themselves. They gradually become more and more feeble mentally; they may cease to keep themselves clean or to attend to their bodily needs. In the end they may need more care than a new-born baby.

EFFECTS OF TOXINS ON THE BRAIN.—A state of intoxication is most commonly produced by alcohol, but this is by no means the only substance producing a toxic effect. Many drugs, especially if absorbed over a long period, will do much the same. In starvation or dietary deficiency patients manifest very similar symptoms. It is probable that some of the symptoms common to all these conditions

are due to lack of the Vitamin B complex. The less chronic cases respond rapidly to massive doses of the Vitamin B complex.

Alcohol has a number of mental effects. Many people find that it makes them feel cheerful and uninhibited. If they go on drinking they gradually lose all control until finally they become unconscious. Next day they suffer from a "hang-over". If they become chronic alcoholics, their character and behaviour will deteriorate and they may reach a stage of dementia quite early in life. Those whose consumption is still greater may suffer from *Delirium tremens*, a condition in which they believe they are seeing mice, rats or other small rapidly moving objects and become panic-stricken. To escape them the victims will do almost anything; e.g. jump out of a window or run in front of on-coming traffic.

DISTURBANCES OF BLOOD CHEMISTRY.--Chemical disturbances, such as those of blood sugar level, of water balance, of acid-alkali ratio in the blood, and insufficiency of oxygen from any cause, all give rise to mental disturbances. In severe illnesses in which the blood chemistry is affected, such mental states may occur as *hallucinations* (Latin *allucinare* = to wander in the mind, nowadays used to mean a false perception), delirium, illusions (Latin, *illudere* = to play false, used to mean a *mistaken* perception). The patient's mood may also be affected, e.g. he may become irritable or touchy, inappropriately elated, or equally inappropriately depressed.

ENDOCRINE DISTURBANCES.—There is some mental change accompanying most endocrine disturbance (p. 231). One of the most marked and commonly occurring is that accompanying changes in the activity of the sex hormones.

The emotional disturbances in both sexes at puberty and in early adolescence are well known, and known to be connected with the sudden great increase in the output of sex hormones. Many women have a monthly cycle of emotional change and suffer from *pre-menstrual tension*. This condition is believed to be due, at least in part, to the retention of fluid in the brain. The change in the water-retaining capacity of the brain is known to be under the control of the sex hormones.

At the menopause, when the cessation of activity of these glands may be sudden, women may suffer from a variety of disturbances. These range from irritability and tension to instability and depression. There seems to be some connection between the tendency to pre-menstrual tension and the tendency to severe menopausal disturbances, i.e. those most severely affected in the few days before

the menstrual period tend to be those most severely affected at the menopause.

Boys often go through similar adolescent disturbances, but men do not have the same sudden change of endocrine activity as women in their late forties or early fifties. There is nothing in a man's life to compare with the menopause in a woman's. On the whole, they suffer fewer mental changes and fewer "involutional" mental disturbances. Men are not exempt from mental troubles, but those that come from endocrine causes they tend to have later and more mildly than women.

THE BRAIN AND THE MIND

It is not surprising that there should be such mental disturbance, nervous tissue being, as it is, the most sensitive in the body and least able to stand lack of sugar or oxygen, or departure from the normal exchange of gases. We are still, however, only at the stage of guessing how these mental changes are produced or, rather, what is the exact nature of the change that occurs in the brain cell and shows itself by a change of mood, of intelligence or of personality. Knowing, however, that changes of something of the same sort occur both in so-called organic diseases and in the psychoses makes the latter seem a little less inexplicable and a little less terrifying. It also gives some hope that before too long the problem of the causation of mental disturbances will be solved.

Mental Causes

INBORN MAKE-UP.—While everyone knows in a rough way what is meant by "personality," it is not an easy word to define. It is used of mental traits in rather the same way as the word "figure" is used of physical traits—it means the sum total of all the things that make one person distinguishable from another. Naturally not all, or even most, of these things are inborn in a person, but everyone starts life with a collection of qualities inherited from his parents, and just as on the physical side one may be weak and another strong, so on the mental side some people are lucky in what they start off with, others less fortunate. It looks as if tendencies towards certain types of mental disturbance were laid down in the individual's constitution just as are tendencies towards certain types of organic disease, but at present less is known of the inheritance of mental qualities. What we believe is:

- (1) that there is a "constitutional predisposition" to certain types of mental make-up.

- (2) there is such a thing as an inborn *poor* personality make-up and that this produces people who are unable to stand up to stresses and strains.

In many mental illnesses, underlying the collection of odd behaviour that constitutes the "disease", we can detect a particular type of personality that seems to be destined to go that way.

ENVIRONMENT.--A person's environment (surroundings) is, in the widest sense, everything that he comes into contact with, everything that produces his "experience of life". It would take too long to put down all the circumstances and events that may have a decisive effect on mental health. It is sufficient to say that throughout our lives, from every part of the environment we receive on the one hand *satisfactions*, on the other, *frustrations* (Latin, *frustrare* = to disappoint). Every life has its different quantities of satisfaction and frustration and these may come in ways that cannot be understood by an outsider. It is their amount and proportion that determine whether an environment is hard or easy. The harder the environment, other things being equal, the higher the chances of mental ill-health.

What is particularly important is the environment in early childhood. The misfortunes, the incidents which leave permanent scars on the character, occur in early childhood or infancy and the sufferers may no longer remember them. Nevertheless the whole character has been affected for the worse. Without knowing the details in any given case, it can be seen that this is likely to be so from the fact now established that the majority of neurotic patients come from "broken" or unhappy homes. Children brought up in institutions, by step-parents or foster-parents, children of divorced parents or of parents who quarrel, children one of whose parents is unbalanced, have a great handicap at the start of their lives. Ill-treatment in infancy, when the child is weak and his personality undeveloped, has a much more damaging and lasting effect than anything of the same kind later in life. The damage is done to his character rather than to his body, and it need not be by gross cruelty. Neglect, lack of love, harshness and severity produce their permanent effects on a character.

CONFLICT

When a patient is suffering because of internal conflict he is, as it were, two people each wanting to go a different way and both wanting with equal strength.

How does this come about? Certain cases can easily be understood. A soldier in danger wants to do his duty, he also wants to get away. So long as the impulse to do his duty remains uppermost, he carries on; if the urge to run away becomes the stronger, he deserts. If both impulses are of exactly equal strength—what happens? The answer is that the state of tension in time becomes unbearable and he breaks down.

Situations of this sort accounted for a great many psychological casualties in the forces. In the example given the conflict is between the wish to do what his training and understanding tell him is right and the wish to respond as any child or animal would respond in the circumstances.

In every person a conflict between these two parts of the mind—the “socialised” on the one hand and the primitive and instinctive on the other—is going on all the time, and adjustment to ordinary social life is successful so long as the “socialised” part of the mind is winning. If the primitive part wins the resulting behaviour is what we call criminal or delinquent. When the forces are equally balanced there is tension and breakdown.

Psychosis and Neurosis

MAJOR PSYCHOSES

GENERAL.—At present we still lack a full understanding of how these diseases are caused. In many cases we see that a patient whose behaviour is so odd as to deserve the description insane, exhibits only an exaggeration or, as it were, a caricature of ways of doing things that are known to us in everyday life. The more fully mental life is studied, the more it is appreciated that nearly everything observed in insane persons can be observed at some time or in some degree in ordinary people at some period in their path from infancy to old age. In ordinary people, however, these things have to be looked for. They are often not to be found at all in adult life except in so far as hints of them come into dreams, imaginings or inventions. The insane person is in this respect just the opposite of the ordinary person. The odd and queer things are happening most of the time. Moments of ordinary behaviour can be observed, but they have to be looked for rather carefully.

PSYCHOGENIC PSYCHOSES

In the psychoses of psychological origin two main groups may be distinguished; that in which all the mental processes are equally

affected; and that in which one function is primarily affected, and the others, if at all, only secondarily.

SCHIZOPHRENIA

Schizophrenia (Gr. *schizein* = to split, *phren* = mind) affects the young more than the old, men more than women. It tends to occur in those who already have a schizoid personality, i.e. in the withdrawn, silent type with whom it is difficult to make a contact.

In schizophrenia thought is *disordered*, affect is *incongruous*, and behaviour is *bizarre*. These three words which are used in the classical description of schizophrenia, all mean the same thing—inappropriate—and are used to convey the idea that the disease affects all aspects of the patient's mental life. This meaning is contained in the word schizophrenia. Although this word is literally translated as "split mind", this does not mean (as is so often thought) neatly divided into two personalities. It means fragmented or shattered, as if the personality were in little bits.

This fragmentation is seen in schizophrenic *thought disorder*. In the ordinary sense of the word think, the patient has lost the power of thinking. He may string together sentences which, to the ordinary person, have no connection with one another. "I saw a man so near death that a breeze would give you a cold," is a typical schizophrenic sentence. Often words follow one another in a meaningless way—a word salad—or the patient's talk may be filled with words, sometimes long and complicated, of his own invention—*neologisms*. Examples of neologisms are "bethrowal" (? a telescoping of betrothal and betrayal), "confusitize" (confuse + hypnotize?) "monogize", "pligtophobia", "glamical". Other more obscure and longer words often appear in these patients' letters.

Part of the patient's thought disorder may be his *delusions*—beliefs which are false but unshakeable.

INCONGRUITY OF AFFECT means that his moods, feelings and emotions are out of keeping with his circumstances. He may change rapidly, and with no apparent provocation, from complacency and fatuousness to rage and violence. Similarly, he may show no sorrow at the news of a death in the family, but go on to say what he wants for his lunch, or what he is going to do for the rest of the day.

BIZARRE BEHAVIOUR.—Schizophrenics may do completely unexpected things; some of their actions may be merely silly or mischievous, but others may be dangerous either to the patient himself or to others. Every year some murders are committed by schizophrenics. It is revealing—and horrifying—to hear them speak of

their "crimes". They may not have known the person murdered or may have had only a slight acquaintance with him. They will describe the murder in unemotional, monotonous tones, expressing no regret and giving no explanation.

HALLUCINATIONS.—Disorders of perception are a common feature of schizophrenia. Auditory hallucinations, in which the patient hears things that are not there are the commonest type in this illness.

VOICES are heard by most schizophrenics. The voices may say something offensive about the patient or may give him orders. Some murders and some suicides* are committed by schizophrenics at the command of the voices.

PARANOIA (Gr. *para* = beside, *nous* = mind) is a form of schizophrenia in which thought disorder predominates and feelings and behaviour are disturbed to a smaller extent and only secondarily. This illness is characterized by delusions. The typical paranoid delusion is the *delusion of grandeur*. In this the patient believes that he is someone more important than he is; e.g. that he is the embodiment of some great man, living or dead, that he is related to some important person, or that, in his own person, he is grander or cleverer than he really is.

The person who has only a slightly inflated idea of his own worth may seem at first sight less deranged than the florid paranoid whose delusion is clearly absurd. One may smile tolerantly at the person who is continually saying such things as "the chief is entirely dependent on my advice", or "I am one of the few people who has a clear grasp of the purpose of this department", but this type of person may create great havoc among colleagues. He will have no doubt that he is entitled to any job he applies for and, if he fails to get it, he will be convinced that he was turned down because enemies plotted against him. He is unable to believe that anyone else could be more deserving than himself; neither can he believe in bad luck. In any misfortune that befalls him, he sees evidence of a deliberate attempt to injure him. He tends to become suspicious and to read hidden meanings into the most innocent actions of the people around him.

Affective Psychoses

In the affective psychoses the most striking feature is the "swing of mood". The swing may be to inappropriate sadness—depression (Latin *deprimere* = to press down, to lower), or to equally inappropriate cheerfulness—mania (Gr. *mainesthai* = to be mad). No events

* We can only know, of course, about the "unsuccessful" suicides.

in the outside world have any influence on the prevailing mood. It may "come out of the blue" and go just as unexpectedly.

Thought and behaviour are also disturbed, but they are disturbed by the prevailing mood. When the mood changes, thought and behaviour change in the direction that would be expected.

Depression is commoner than mania; it may occur at any age, though it is more a disease of the old than of the young, and extremely rare in children. It tends to recur, and it occurs more frequently in women than in men.

Depressed patients are apathetic (without feeling) and retarded (slowed down). They usually sleep badly, often lose their appetites and become emaciated. Most depressed patients move little, show little facial expression and maintain a sad, unsmiling appearance. Their utterances and thinking are slowed down.

A depressive illness is often associated with a period of physiological stress, e.g. it may be a sequel to infective illness, often complicating influenza or virus encephalitis. It may occur at puberty, at or just after child-birth, and at the "change of life" (or more accurately, in the years before or after the menopause). These *involutional* depressions, as they are called, are of a special character, being marked by severe *agitation*, rather than by apathy and retardation.

Depressed patients may die of malnutrition or of intercurrent (physical) illness, to which their inactivity makes them prone; but the chief danger in this illness is suicide. The mood of depression is unbearable and drives many patients to take their lives. Few of these patients are a danger to others, but in a post-puerperal depression a woman may take not only her own life, but also that of her baby, or even the lives of several children. The risk of suicide (and in the post-puerperal cases, of infanticide) is great, because so often it is the patient with the seemingly mild illness, or the patient who is recovering who makes the suicidal attempt.

MANIA

The manic patient is over-active and over-excited. He hardly stops talking and, as he talks, he winds himself up, one thing leading to another, so that at the end of a few minutes he may have covered all manner of topics in a carefree jovial way. He has what is called "a flight of ideas". In his flight it is always possible to see the connections between one idea and the next, though the last in the chain is so remote from the first that a number of different people might have filled in the intermediate steps.

A manic patient is described as follows: "she is all over the ward, dancing, singing, slapping patients and nurses on the back, pulling off her clothes and throwing things about in absolute abandon. She writes poetry and insists on reading it to everyone near her, monopolises the conversation and has a flippant reply for every remark made by anyone else. . . . She writes page after page of material, the content of which rapidly shifts from one subject to another. She underscores and capitalizes at random."*

TREATMENT OF THE PSYCHOSES

Success in all branches of medicine has depended on an understanding of the causation of the malady. This is as true of mental as of physical illnesses, and, unfortunately, there is still much to be learnt about the causes of mental disease. The basic principles of general treatment are the same in all illness (see p. 1). The first essential is that the patient should rest in mind and in body. Hence the psychotic in the acute stage of his illness should be in hospital. He may be in poor bodily health and, on that account, need rest and nursing care; but what every patient will need is rest for his mind and freedom from responsibility. This he can only get in a specialised hospital.

ORGANIC PSYCHOSES.—When the cause is organic, ridding the patient of the damaging causative agent is indicated, whenever this is possible. In syphilitic and alcoholic disease, for instance, elimination of the injurious organism or toxin has given good results. Unfortunately, lost brain tissue cannot be replaced and the patient may be left with a permanent disability. Arrest of the destructive process, with re-education of the remaining capacities, is the best that can be done.

In mental disturbance due to generalized brain deterioration there is even less hope of restoration of function. Nevertheless, perseverance in re-education with attention to every small impairment of function will do a good deal to make these people healthier and happier. Elderly patients are often greatly helped by *tranquillising* (Latin, *tranquillus* = free from agitation, calm) drugs. The senile patient may be able to take drugs of this type in very large doses without side effects, and are often almost miraculously freed from agitation and restlessness.

PSYCHOLOGICAL PSYCHOSES.—Our lack of understanding of the

* Dorcas and Shaffer. *Abnormal Psychology*, 1934.

causation of these diseases leaves us without any radical or infallible cure.

Again, rest and general measures play a great part in treatment. The chronic patient, in particular, gets help from an environment which, without making too many demands, is nevertheless stimulating and interesting. Most mental hospitals try and get as many patients as possible to work for part of the day, to take up "occupational therapy", to join social clubs where they have an opportunity to take part in games and dances, play-reading, acting, music and painting. The internal organisation of hospital and hospital wards aims at raising morale and getting the patients' active co-operation in the running of their own day to day affairs.

PHYSICAL TREATMENTS.—Some of the depressive illnesses respond very well to *electro-convulsant* therapy (E.C.T.). The patient is treated by the passage of an electric current through his brain. A series of eight or ten, or even more treatments is usually given and may have to be repeated at longer or shorter intervals. It is particularly effective in the treatment of the "agitated" depressions of the menopausal woman.

In recent months many drugs have been tried in the treatment of depression, in the hope that a substitute for E.C.T. might be found. This hope has not been, so far, completely realized (though it may be near at hand) but a number of drugs are now available which, used in conjunction with E.C.T. speed up the treatment in many cases and make relapses far less likely than in the past.

Schizophrenia, more particularly the acute attack in a young person, is often treated by *insulin coma* therapy. The patient is given an injection of insulin sufficient to produce coma, and the coma is then reversed by the injection of glucose. It is a procedure attended by obvious dangers which should only be carried out in a specialized unit by a team thoroughly trained to undertake it. This treatment, too, can now be supplemented by drug therapy. There are numerous drugs now that give excellent results in the management of chronic schizophrenic patients. There are now few patients who cannot be helped to some extent, even if they are not completely cured.

Special Problems of the Psychoses

A psychotic patient may be in hospital not only for treatment, but also for custodial (guarding) care because of the likelihood that he will be a danger to himself or to others. Hence, the care of the psychotic patient is a legal as well as a medical matter.

To protect the public, to give the patient his rights and to treat

his disease may call for courses of action that the hospital does not find easy to reconcile with one another. Up to the present, therefore, rules for the guardianship of mental patients have been the object of Acts of Parliament. It will always be necessary to interfere with the liberty of some psychotic patients, but experience has shown that many regulations formerly in force were unnecessary. The most recent Mental Health Act aims to remove as many restraints as possible, and, except to violent and irrational patients, to make the mental hospitals as open as any general hospital, and admission and discharge as informal.

Another group of patients in mental hospitals who need special provision are the long-term patients who have ceased to be seriously ill, but who are not fit to lead an independent existence in the outside world. A good mental hospital provides sheltered employment and sheltered social life for the permanently inadequate. It is thought that, as a result of bad hospital care in the past, some patients have become "institutionalised" and that there should be fewer in the future. Though it will always be necessary to provide some care that is social rather than medical, the present aim is to make and to keep the hospital a place for medical treatment of medical conditions.

NEUROSES

Neuroses are those disorders of mind in which the patient makes reactions that are inappropriate, not at random or accidentally but regularly in certain situations. Mental functioning is interfered with, but not completely. Nor is the patient, as in the psychoses, completely out of touch with reality. Neurotics may have some understanding of their disturbance, without being able to control it or alter it.

Neurotics endure great mental suffering and should not be regarded as weaklings who do not try to "pull themselves together". Many are highly courageous people struggling against great odds. They may be predisposed to their disorder by their inborn make-up, but they are also people who have experienced great strain and hardship.

Stresses may come from the non-human environment, from the human environment or from conflict between impulses within the person himself. These stresses may be called

- (1) impersonal,
- (2) inter-personal,
- (3) intra-personal.

Examples of impersonal stresses are exposure to cold, to hunger, to danger—a soldier extricated from a blazing tank in which he believes himself trapped will often be a neurotic. He may have been the bravest soldier in the army, but without suitable treatment, he will remain a neurotic, i.e. a mental cripple, all his life.

Inter-personal stresses occur to some degree in all human organisations and in all families. They do not produce neuroses unless they are severe, persistent and inescapable. The patients seen by psychiatrists are often the victims of homes that have never been happy and of parents who have been in perpetual conflict with one another.

The victim of the inter-personal stresses of a bad home will be almost bound to develop intra-personal conflicts. These conflicts often arise in patients who have had quite a reasonable home background. In any human being, no matter how he has been brought up, there will be some conflicts between impulses. For example, a young woman who has a strong craving for sweets and, at the same time, a strong desire to slim will gain or lose weight according as one or other of these wishes is the stronger. If, however, the two wishes are equally balanced, she will find herself in the throes of a conflict. This comparatively minor intra-personal conflict is of the sort that most people experience from time to time but are usually able to solve for themselves.

ANXIETY

Anxiety and fear are closely allied; the term *anxiety* being used to describe the feelings accompanying the anticipation of some ordeal or dreaded event, the term *fear* to describe the feelings that come from being face to face with it. In war soldiers experience both fear and anxiety, and many say that anxiety is worse than fear. In peace the ordeals are not, for the most part, so severe, but students waiting for an examination and patients waiting for an operation feel that the ordeal is bad enough. The anxiety that they and the soldiers feel is "normal" anxiety. It is an unpleasant feeling, but in anticipation of an unpleasant event.

The worst feature of anxiety is the "mental anguish", but it is accompanied by physical symptoms which are themselves unpleasant.

Fear makes us tremble, it makes the legs weak, the skin pale and clammy, it makes the heart palpitate and the breath come fast and painfully. Anxiety may do all these things, continuously or in attacks.

Neurotic patients may feel the mental anguish or the physical symptoms, or both, without the threat of a recognizable ordeal. They may feel anxious all the time (chronic anxiety state) or they may

have attacks (acute anxiety state). Some feel anxiety when shut in anywhere, e.g. in a cinema or an underground; some when they are left alone, some in the dark: some are made anxious by heights, some by any form of transport; and some cannot even go shopping or out into the street.

Patients often complain of waking in the night with their heart racing and with the feeling that they cannot breathe or that they are going to choke. They may have severe pain in the chest, feel giddy, feel their legs give way under them, suffer from distressing frequency of micturition or from diarrhoea. The patient develops a secondary state of anxiety because of his symptoms, and a vicious circle is set up. He interprets the disturbances in his chest as heart disease; the other symptoms suggest cancer to him.

These patients show no relief or pleasure when they are examined and assured that they have no organic disease. They are often very difficult to treat because they are genuinely unaware of the reason for their anxieties and fears. In treating them it is assumed that their anxiety and fear always have a cause, though the cause may be something that happened many years before they began to feel the symptoms. If a cause can be found which the patients can understand and accept, they usually lose their symptoms.

HYSTERIA AND ALLIED STATES

In ordinary speech a "fit of hysterics" means an uncontrollable outburst of laughing and crying, but in psychiatry the word hysteria has a specialised meaning. It comes from the Greek word *Hystéra* (= uterus). The Greeks thought that the uterus moved about the body and that, if it wandered too far, it caused a disturbance of the mind.

Consider again the state of mind of the soldier kept in a position of danger, not by being trapped, but by a sense of duty. At some point the wish to run away and the wish to remain at his post may be equally strong. Here is a case of an irresistible force meeting an immovable object. Only a major disaster can resolve this conflict. What happens is a severe mental illness.

Many conflicts in civilised people are of this order. The wish to behave well in a democratic group may be equally strongly opposed by the wish to have one's way. Hatred of the winner may balance the wish to be a good loser; fury at parents may be equal to the wish to honour them. A man's disapproval of himself may be equal and opposite to his sexual desire, or to his covetousness.

It is the opposition of two equal or nearly equal forces that leads

to the type of disturbance termed *hysterical*. The soldier torn between duty and instinct may lose the use of his limbs, his sight, his speech, his memory. The *functions* of walking, seeing, talking, etc., will go, although no pathological change can be detected in muscle, nerve or sense-organ. Similarly the man with a sexual conflict may become impotent; the child torn between aggression and obedience may begin to stammer or to have attacks of asthma.

Various names are given to this sort of neurosis. It is sometimes called conversion hysteria, because a mental disturbance is *converted* into a physical disability. It is also called a *functional* illness because only function and not structure is affected. This is not a very good name, because, whatever may underlie the illness, if function is disturbed for long enough, structure will change. Thus, it is now considered that neurotic conflict plays a part in the causation of such diseases as peptic ulcer, colitis, migraine, skin rashes and many others. Since different people react with disorders of different organs or bodily systems, it seems reasonable to suppose that innate "constitution" plays some part in the causation of disorders of this type. Hence they may be described as *psycho-somatic* (Gr. *psyche* = mind, *soma* = body) disorders. This seems, for the present at least, to be the most satisfactory term to use.

Hysterical, though much used to describe this type of disorder, is perhaps better kept as a descriptive term for a certain kind of personality. The hysterical *person* is "unduly responsive" to the situation in which he finds himself; the responsiveness being the outcome of an immature, childish dependent attitude to people. He remains a child, with a child's selfishness, self-centredness and irresponsibility. He is very suggestible and is likely to be prone to some or several psycho-somatic illnesses.

Those who succumb to psycho-somatic illnesses are sometimes said to "gain through illness". There are few people who do not know what it is to "gain" through illness, whether it be the "convenient" illness that provides an excuse, or the real but minor illness that makes it impossible to keep a disagreeable engagement, but does not interfere with a pleasant one. The neurotic's disability goes further than this and gets him out of unpleasant commitments for an indefinite period. But just as the normal person does not get a cold or a headache on purpose, the hysteric has no conscious intention to produce his symptom. He could not, by an act of will, paralyse a limb or deprive himself of speech, sight or hearing. What is more, never to be able to walk, to see or hear or to use one's voice is a high price to pay for the "gain". As we have seen, what the patient does is to

substitute a bad but tolerable condition for an intolerable and insoluble conflict.

OBSESSIONAL NEUROSES

By an obsession (Latin *obsedere* = to besiege, beset) is meant an inescapable preoccupation. Many people have an obsessional personality, i.e. they tend to be over-conscientious or, when they are better understood, to be self-driving and self-punishing. They are afflicted by *compulsions* or by *doubts* or by both.

In the obsessional neuroses these traits are exaggerated to the point of severe suffering. The compulsion may be an idea or an image that the mind cannot get rid of. It may be an urge to do or say something that becomes the more irresistible the more the patient tries to resist. If the impulse or image is frightening, the patient is said to have a *phobia* (Gr. = fear, dread), which may be attached to something which to others is harmless (e.g. cats, mice, flies, spiders).

The obsessional tends to exaggerated scruples about conduct, to extremes of cleanliness and tidiness. It is a positive advantage to be neat and tidy; and it is not abnormal to dislike cats or spiders. It is only when these things interfere seriously with ordinary life and with the patient's happiness they cross the border and become neurotic symptoms.

Doubt and indecision are the outcome of the inner preoccupation, since it is continually distracting attention from his conscious problems and does not give the patient a chance to think them out. This state of inner distraction is one of the most painful features of the illness. It accounts for such abnormal behaviour as going back again over a column of figures and rechecking no matter how many times it has been checked already, of returning home again and again to make sure that the lights are turned off or the doors locked, and remaining uncertain about these things no matter how certain everyone else may be.

In suitably chosen occupations, patients whose obsessions are not too severe do quite well, but only so long as no new adaptation is called for. Mr. X worked in a grocer's shop in a small town. He was often late, because he took such a long time dressing himself and cleaning his house. If he omitted any item in his cleaning ritual, he felt a compulsion to start from the beginning again, and often did so six or seven times. To compensate for his unpunctuality, he was the most conscientious worker in the shop and never minded working late or in the week-end to get some job finished and quite perfect. His employer accepted him with all his oddities, and all went well for

many years until the business changed hands and clocking-in was instituted. Mr. X made a desperate effort to be punctual, but found it harder and harder to get through his self-imposed cleaning programmes. Soon, he gave up his job, and a little later, he needed mental hospital treatment.

TREATMENT OF THE NEUROSES.—In the neuroses the assumption is that the causes, or the most important causes, are psychological. In many cases psychological treatment is effective. It ranges from common sense talking over difficulties to the elaborate technique of psycho-analysis in which an attempt is made to get back to events of the patient's earliest infancy.

If, e.g. the girl in the conflict between slimming and self-indulgence sought psychiatric help, the psychiatrist would set out to help her understand why the conflict was so insoluble. Then, according as he thought it important for her to lose or gain weight, he would try to help her to decide on the best course of action and to stick to it.

If he were dealing with the soldier trapped in the burning tank, he might find that the patient had "forgotten" the incident. As can readily be believed, an experience such as this is too fraught with emotion really to be forgotten. It has not gone *out* of the patient's mind, but into the bottom of his mind, and he has so pressed it down (*repressed*) that, unaided, he cannot get it up to the surface. Nevertheless, it is still causing trouble as are the efforts to keep it repressed. Peace of mind will come only when he has recovered it. In cases of this sort, the technique known as *ab*-reaction (Latin *ab* = off, away from) is employed. The patient is lightly anæsthetised and then stimulated to live through the experience. Several treatments may be necessary, but eventually, the experience will become conscious and will be remembered. Once this has happened, it will begin to lose its terror for the patient.

The patient who uses an illness to extricate himself from an intolerable situation may be suffering from a heavy burden of guilt. His problem is to regain his self-esteem. Ordinary consolation and re-assurance will not help him: he does not compare himself with other people and gets no comfort from knowing that he did better than most others would have. He may be judging himself by some impossibly high standard. Before he can be helped, it is necessary to know why he has this standard for himself. To find this out, it may be necessary to go back over the greater part of his life, spending an hour a day for five days a week for several years in doing so.

The object of treatment may be to rid the patient of one conflict,

it may be to solve a number of conflicts and so lessen the need for repression. More ambitious treatment may aim at a thorough-going reorganisation of the whole personality, adapting it better to the world the patient is living in, and getting him directed to what is going on in the present instead of being fruitlessly taken up with what has happened in the past.

Note on Treatment of Psychotic Patients in a General Hospital

Most psychotic patients will be very difficult and unsuitable for treatment in an ordinary ward. Some may need nursing in cot beds with unremitting supervision and heavy sedation. Feeding is a great problem for they may have to be hand-fed or tube-fed. The important thing for the ordinary nurse to remember is that patients in these states are *irrational*, that they will not co-operate and they will not be influenced by any of the means which influence an ordinary patient. It is quite useless to argue with them. This is particularly true of paranoid patients, but a temptation that it is often difficult to resist. Paranoid patients often seem more than usually intelligent. They express themselves well and try to draw other people into arguments. When their beliefs seem quite preposterous, it is difficult to refrain from telling them so. If they insist on saying that all the food in the ward is poisoned, the nurse's first tendency is to deny this with indignation. She can deny and she can prove her point in a way which would satisfy any jury, but she will not have the slightest effect on the patient. If her aim is to get him to have something to eat, it is sometimes possible to get round this sort of difficulty by a little cunning. Leading the patient on, one may get him to indicate that there is some person whom he trusts; his wife or his mother or his sister, it may be, does not poison his food, though everyone else in the world does so. With a little cleverness he can sometimes be convinced that his food is brought to him by this person. It is only a short-term measure, but so is his stay in the general hospital.

If a severely disturbed mental patient turns against a particular nurse, it is much better for that nurse to have nothing more to do with him and let some other take over. While one nurse is everything that is bad, some other nurse in the ward may appeal to the patient as having "the voice of an angel" and every perfection. It simplifies life for all if, while this lasts, the angelic nurse is the one that looks after him. He is equally likely to turn against her at the end of a few days, but it may contribute to relative peace until he gets into the mental hospital.

The Psychology of the Ill Patient

FEAR AND ANXIETY

It is very easy for the workers in a hospital to forget what coming to hospital means to the patient. Most patients will not have been in hospital before, and the mere fact of being sent to hospital shows that they are gravely ill. They are, therefore, bound to be afraid. It is not easy to rid them of their fears, and mere assurances that everything is all right and that they have nothing to worry about will not help them very much. What they need is to be shown that the people in charge of them understand how they are feeling and sympathise with their anxiety, although they may want them to overcome it. Patients' fears about their own health and about the prospect before them in hospital can be added to by fears about what is going on in the outside world which they have left. A woman may be agitated and worried that her home and children will not be properly looked after. A man may worry desperately about his job and his income.

CHILDISHNESS

The ill patient, particularly when he has gained some confidence in his nurses, is apt to give up some of his independence and willingness to stand on his own feet. He is, after all, no longer on his feet. For many purposes he has to be waited on, and it is easy to go back to the state of mind of taking for granted that he is the centre of the universe and that everyone else revolves around him. At some part of any severe illness the patient is like a child, and of necessity is being treated like one. If the illness is long he may take on this attitude permanently, become very demanding and self-centred and find that continuing to be ill holds out a more agreeable promise than making an effort to get well.

Here we have two emotional reactions which will probably be brought out in every illness, namely, fear and a tendency to become childish and self-centred.

HELP THAT THE NURSE CAN GIVE

The nurse who knows that her patients will react in these ways can give them a great deal of help. She can give them a chance to talk about their fears, to voice their anxieties about things which are or may be going wrong in their homes, and she can prevent herself and other members of the hospital staff from doing and saying things which add to their anxieties. Medical discussions at the bedside very often leave the patient in a state of agitation because he has not understood the whole discussion, or grasped the conclusion reached

by the doctors, but has picked up some word or phrase which has for him an alarming meaning. On the other hand, the habit that some physicians have of discussing at the bedside the less severe cases and then pointedly walking away from those who are seriously ill to talk about their case at the other end of the ward does nothing to reassure the anxious patient. Most patients tell one that they want to know more about their own condition than their medical attendants ever tell them. Obviously the nurse is in a difficult position here. If the doctor is deliberately keeping information from a patient, she must not impart it, but when a doctor has told the patient about his condition, but too briefly to make himself plain, the nurse can help a great deal by explaining in simple language what it was that the doctor really meant.

A tendency to childishness needs a rather special effort from the nurse. On the one hand it makes the nurse's task easier for her. The patient in a way plays into her hands by putting himself into the role of child, and her into the role of mother. On the other hand in his "childishness" he will exhibit the unaccountable obstinacy of a young child, the fretfulness and peevishness and the inability to put up with waiting and postponement that most young children show. This sort of behaviour is very irritating to a nurse. She is apt to be flummoxed by an adult behaving in a tiresome and unreasonable way.

INCONTINENCE

Many patients will show forms of childish behaviour which are even more trying. The elderly patient is, as we know, apt to be incontinent. Most adults tend to be disgusted by this lapse. In a ward set aside for senile patients nurses often show a tolerance and sympathy which is beyond all praise, but they do not always realise that these attitudes may be called for in an ordinary ward. A case may be quoted. A young woman in her late 20's after an operation had both legs put into a plaster which was carried up to the waist. A few days after the operation she was given an aperient but when she asked for a bed pan was kept waiting. The plaster and the position in which she was lying made muscular control extremely difficult and she was incontinent of fæces. The nurse in charge turned on her with every expression of disgust and horror: "You, a grown woman, should be ashamed of yourself." The patient *was* ashamed of herself. She was completely horrified and distressed that such an accident could have occurred. The rebukes of the nurse completely unnerved

her and she had a very unhappy time in the ward for the remainder of her stay.

The nurse should be able to tolerate incidents like this without herself becoming emotional. In this particular case she should have known that it was quite beyond the patient's control, and should have known how distressed the patient would be because of her own lapse. This was an occasion when the best reaction to the patient's childishness would have been to behave like the mother of a young baby and either clear up the mess without any fuss or comment, or, better still, with some word of comfort to the patient, assuring her that it was the result of special circumstances and most unlikely ever to occur again.

The Neurotic Patient in Hospital

What has been said of the ordinary patient in hospital applies with even more force to the neurotic. "Neurotic" is a word which nearly everyone uses nowadays, and more or less understands, but it is hard to make its meaning precise. It might be said that neurotic persons are unhappy persons, though the converse is not true; people can be unhappy without being neurotic when the cause for the unhappiness is obvious.

The fact that there seems to be no obvious cause for their unhappiness makes it difficult for us to feel sympathetic towards them or to be patient or tolerant. They are making a constant demand for our sympathy and yet they cannot produce any reason why we should give it to them. In an ordinary ward, the great majority of the patients, in spite of their troubles, are well behaved, co-operative and considerate. They can see for themselves how busy the nurses are, and they can note that, if a nurse is not attending to them when they want her to, it is because she is already occupied in attending to someone else. The neurotic patient is incapable of seeing things in this light. If he wants attention, he wants it now and he cannot make allowances for not getting it. He cannot see that if he merely wants a drink of water while someone at the other side of the room is vomiting or bleeding, he must wait until the urgent crisis has been dealt with. If he has any pain, he makes a great fuss about it. If he cannot sleep, he is full of self-pity. If he does not like the food, he complains and may lose his temper. He is, in fact, the trying or difficult patient, one who seems to set out to make the nurse's task harder instead of, as the majority do, trying to meet her half-way.

It is not easy to be kind to this sort of patient. In the first place, he wants more than his share of attention. Further than that, he

seems to bring out the worst in us in that we are all rather apt to react by wanting to give him less than his fair share of attention because he is asking for too much. It must be agreed that a patient of this sort is less likeable than the brave, uncomplaining patient who puts up with pain and discomfort as far as he must, and who can make a joke and cheer up his fellow sufferers and take some of the burden off the nursing staff.

It is, however, the job of the nurse to care for a patient just because he is ill and not because she likes him or finds him easy to deal with. One of her reasons for being rather harsh with the neurotic patient is that she feels that he is making a lot of fuss about nothing. If she realises that it is not, as a matter of fact, about nothing, it may be that she will be less irritated and provoked by his goings-on.

Nurses and doctors have a rough and ready idea of how much pain is bearable and where it becomes too bad to be endured. At that point we give people analgesic drugs or anæsthetics. Probably our assumptions are fairly correct and when we say that someone is "making a lot of fuss" in most cases his pain is not very severe. But the real trouble is that he is both pained and frightened and the combination is too much for him to stand. The question then is, why are some people so much more frightened than others, or why are some people so much better at hiding their fear? The answer generally is that life has treated the neurotic patient worse than other people.

Sometimes a neurotic patient who is given a chance to tell his story, immediately rouses our sympathy and understanding. For example, a sergeant of 24 in the A.T.S. broke down and had screaming fits. On getting her confidence her story was that she had recently discovered the man she was engaged to was married already, that six months before her father had died of a cerebral hæmorrhage and she had been alone with him at the time of his death, that for the whole of her life her mother had been a drunkard, who had ill-treated her in childhood and frequently brought shame on the family by spending all the housekeeping money on drink and being charged with assault. It is only necessary to know the facts in a case like this to feel sympathy with the patient and to accept and make excuses for her behaviour. It is not so simple to understand what is upsetting most neurotics, because very often the patients themselves do not know.

Children in Hospital

Fewer children are now being admitted to hospital than ever

before. This is partly because there are fewer serious diseases and partly because many hospitals have developed facilities for treating children in their own homes. Even when children are admitted they are sent home again much earlier than in the past.

Nevertheless, many hundreds of children are admitted every year and most of them will not only be coming to hospital for the first time but for the first time enduring separation from home and mother. Nothing is more pathetic—and nothing more significant—than the chorus “Mummy, mummy, mummy” that can be heard at almost any hour in almost any children’s ward. In pain or in terror, the child’s greatest need is for his mother, but he is deprived of her in what is probably the worst crisis of his young life.

Almost all hospitals now allow children to be visited daily. This means a great deal to a small child who may feel that he has been handed over for ever to strange and hostile people. One London hospital arranges for the mother to come in the evening to give her child his supper, wash him and get him ready for the night and then stay with him till he is asleep.

In Newcastle the late Professor of Child Health, Sir James Spence, organised his children’s ward into rooms in which the mother could stay with her baby throughout his illness. He felt that this robbed coming into hospital of many of its terrors for the small child, brought out the best in the mothers, and made it possible for them to learn procedures that would be necessary to the care of the child after his discharge. The nursing staff also benefited by being freed from all the jobs that the mothers could do, and left time to concentrate on the skilled professional tasks which only they could perform.

PROCEDURES THAT CAUSE FEAR

Children are frightened of hospital procedures. Many are terrified of injections. The pain of an injection is trifling, not greater than that the child must experience many times in his ordinary day. It may seem silly to “make such a fuss.” It is, however, no use our coming to this conclusion, if the child has not. It is our business to observe that this procedure does cause pain and fright to a number of our patients and to think out ways of making it less of an ordeal to them. Similarly, many children resist having their throats examined. The examination may be conducted only after the child has kicked and screamed and been held down by one or more adults. The tonsils are seen but what has been done to the child’s mind in the process?

In a busy out-patients’ department, it is difficult to avoid an occasional act of brutality of this sort, but in a ward where there is a

little time to spare, it is wonderful what can be done by gaining the child's confidence and preparing him for what is going to happen.

Nurses should also realise the particular terror that the introduction of enemas or urethral catheters is likely to produce in children. One case remains in my mind of a six-year-old girl who put up a very violent struggle against catheterisation and was stigmatised as thoroughly naughty and held with great force by the staff. She later revealed that only a few weeks before she had been the victim of an indecent assault and that the catheterisation to her was a repetition of this appalling incident.

FEEDING DIFFICULTIES

Feeding of children is another matter on which the nurse should cultivate sympathy and imagination based on knowledge of the significance of food in a young child's life. Many adult patients have described the horror they still feel at the memory of having food forced down them in hospital or convalescent home.

CHILDREN'S ATTITUDE TO ILLNESS

One other point is what children think about their illnesses. Like the adult hysteric, the immature mind of the child is "excessively responsive." They respond to our moods, our gestures, our movements, partly because we do not give them clearly expressed information to respond to. They have to cultivate sensitivity to the unsaid for lack of saying. They draw their own conclusions, which may be right or may be wrong, but are often very frightening. Thus, one little eight-year-old cardiac patient, referred to a Child Guidance Clinic because of nightmares and sleep-walking, said he thought he might fall down dead. Why did he think this? Because his heart was "bad." What was a bad heart? It appeared that to him it was like a bit of bad meat—a horrible thing to have inside one. An hour's talk on what the heart was like, what it did, what the "badness" (mild mitral stenosis) consisted of, telling him in language suited to his age that "a heart is what a heart can do" and a discussion with him and his mother planning out what he really could do left him a transformed child. This bit of "re-education" was undertaken by a psychiatrist, and perhaps at the stage he had reached it had to be done by a psychiatrist, but he need never have reached that stage. Months of misery for the child and anxious worry for the mother need never have been endured if he had been properly handled when he was first ill.

Bad handling over these items of the daily routine can do lasting

and often irreversible harm to the minds and personalities of growing human beings. It can be avoided by the use of thought, care and imagination.

When a sick child is before us our own five senses tell us a great deal about what is wrong with his body; we know immediately what to do and what to avoid to supply his physical needs. We do not know and cannot know in the same way what is wrong with his mind. When we do not know what his life has been, we should not assume that nothing is wrong. If he is difficult, we should believe it is because he has been through difficulties.

When the patient is in hospital he is in our power. The possibility of accidentally doing harm to his body is never far from our thoughts; our own professional consciences, the routine and precautions of our hospitals are all directed to making such accidents non-existent. It is not nearly so forcibly impressed on us that we have an equal power to do accidental injury to the mind—the mind which we all believe to be more important than the body. Doctors and psychologists as well as nurses still have much to learn about the mind, and the more we know the more surely shall we be able to help our patients. For in our profession, ignorance is not bliss but danger. The first thing we need to know is how little we know; the second and most important is not to rush in where angels fear to tread.

APPENDIX A

SOME PRACTICAL PROCEDURES

A NURSE's job is largely a practical one. In this section a few practical tips are given about procedures in which both doctors and nurses are concerned. The larger part of a nurse's practical work is not described, for a doctor is not qualified to write about such matters. Although technique is stressed in the following paragraphs, I am quite aware that a perfect technician may be a very bad nurse. A patient having a blood transfusion or lumbar puncture is not just a lay figure, and a calm, confident, considerate nurse may make all the difference to his state of mind on these occasions—and, incidentally, to the state of mind of the doctor performing the operation.

(1) Injections

Injections are given when the drug, or whatever it may be, would be destroyed in the gut if swallowed (as with insulin), if the speediest action of the drug is necessary (as with intravenous injections of digoxin), or if the patient is vomiting.

Patients often judge a nurse on her skill in giving a painless injection. The first secret of success is having a *sharp needle*. Insist on discarding blunt needles and those of fish-hook type, which catch up a wisp of cotton-wool from a swab drawn across the point. Always make all ready for an injection before approaching the patient, decide on the site of injection, arrange everything to your satisfaction and the patient's comfort, cleanse the skin and give the injection *quickly* and without hesitation or fuss. Your reward will be the patient's surprised, "Why, I hardly felt *that* one, nurse."

Intradermal injections are used mainly in skin tests, such as the Mantoux (tuberculin) and Schick (diphtheria susceptibility) tests. A minute amount of solution (0.1 ml.) is injected into the most superficial layers of the skin. Insert the needle obliquely so that the point is only just buried, and inject the fluid, which will raise a little white button on the skin.

Hypodermic (subcutaneous) injections are widely used, and nurses are probably more familiar with the technique than doctors. Any drug which is not too irritating to the superficial tissues can be given

in this way; large hypodermic injections are uncomfortable, and, as a rule, not more than 2 ml. of fluid is given by this route. Pick up a fold of skin—for instance, over the forearm—and inject obliquely into the fold.

Intramuscular injections are used when the drug is too irritating to be injected under the skin, or when more rapid absorption is required,

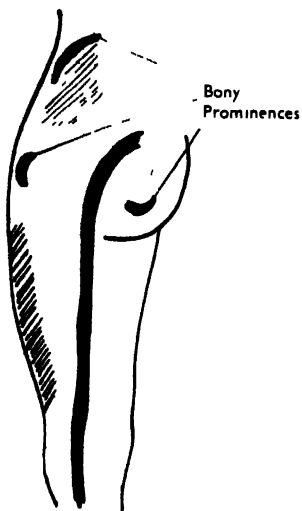


Fig. 107. Intramuscular Injections. Diagram to show the course of the sciatic nerve in the thigh. Safe sites for intramuscular injection are shaded

for drugs are absorbed more rapidly from muscle than from subcutaneous tissue. Give the injection into a thick muscle, such as those of the outer part of the upper arm, the outer part of the thigh or the buttock. Avoid (a) places where you might injure important structures (many people have been given sciatica by being given an injection into the sciatic nerve) and (b) places which will cause the patient discomfort (in giving an injection into the buttock, avoid the part the patient sits on). The diagram shows where the sciatic nerve runs; you can see that the outer part of the thigh and the upper and outer part of the buttock are quite safe for your intramuscular injection.

When giving an injection into a muscle hold the skin taut with the left finger and thumb and inject deeply, but never bury the needle up to the hilt—it may break off at this point, leaving the whole shaft buried in the patient's flesh, whence it may be almost impossible to extract it.

Intravenous injections are used when very irritating drugs have to be injected (such as aminophylline) and when the most rapid action is required, for the drug, being put straight into the blood stream, can get to work without delay. Thus intravenous digoxin is used in acute heart failure, and intravenous insulin in severe diabetic coma. Intravenous injections are usually given by doctors.

(2) Examining the Throat and Throat Swabbing

The most important part of this undertaking is getting a good view of the throat without making the patient gag. With a child, you will need an assistant. The child sits on her knee leaning back against her chest; she puts one hand on his forehead and with the other holds

both his hands. A hand on the child's hands and one on his forehead are also needed with the child in bed. If possible have the patient facing a window—this is much better than fiddling about with a torch. Now ask him to open his mouth and say "Ah." You probably won't be able to see the tonsils and back of the pharynx without using a spatula or the handle of a spoon (I prefer a dessert spoon) but get the light and patient's head adjusted at this stage so that the light falls on what you can already see of the back of the throat. Then the *moment* you hold down the tongue with the spoon you will be able to see what you want without any more fiddling about, before the patient has time to start retching. In *swabbing the throat* rub the end of the swab *quickly* over each tonsil and the back of the throat and withdraw without touching the palate or tongue, which would make the patient retch and would contaminate the swab.

(3) Ear Syringing

Ears are syringed to get rid of wax which is annoying the patient by making him deaf, or the doctor by preventing him from examining the drum. If you are to be a successful syringer of ears you should be able to use an auriscope yourself, for you will never know if all the wax is out unless you can look and see. To examine the ear drum, pull the patient's ear back and insert the speculum of the auriscope in the direction of the passage—forwards. You can recognise the drum by its shiny, greyish appearance—quite unlike the pink wall of the ear passage—and by the white knob in the middle, formed by the protrusion of one of the minute bones inside the middle ear.

When syringing the ear, have the patient sitting with that side towards the light. Swathe him in towels and mackintosh sheets, and give him a kidney dish to hold against his neck to catch the drips. Get him to lean his head towards the side you are syringing. Use warm water, saline or sodium bicarbonate solution. Fill the syringe with your solution, get rid of air, and insert the nozzle of the syringe just inside the ear hole. Hold it firmly against the roof of the passage and syringe *gently*. From time to time examine to see if you can see the drum; if you can the wax is all out. If no wax comes out after five or six syringefuls it is useless to go on; ask for help or tell the patient to use warm olive oil or bicarbonate drops for a few days and then try again. The wax may now be soft enough to come out quite easily. *Never* use any force when syringing an ear, or you may break the ear drum.

(4) Lumbar Puncture

A lumbar puncture is done when a specimen of cerebrospinal fluid is wanted for examination, or when a drug, such as streptomycin, has to be given into the spinal canal. Surgical asepsis is necessary when doing a lumbar puncture and all needles, syringes and other apparatus must be autoclaved. Of great importance when assisting at a lumbar puncture is to get the patient into the right position. The spine must be bent forwards as much as possible, because this opens out the vertebræ and allows the lumbar puncture needle to enter the spinal canal between two vertebræ. The patient lies curled up on his side with his knees drawn up as far as possible. The shoulders and hips should be at right angles to the bed, not slanting. Having prepared the trolley and draped the patient with sterile towels, leaving the lower part of the back exposed, go round to the other side of the bed so that you can hold the patient still if he is restless. You can control him perfectly by putting one arm under his drawn-up knees and the other round his *shoulders*—*not* the neck, because it hurts the patient to pull his head forward, especially if he has meningitis. The patient in position, the doctor sterilises the skin and inserts the lumbar puncture needle between two vertebræ—generally the 4th and 5th lumbar. On withdrawing the trocar C.S.F. drips out if the needle is in the right place. A manometer is now attached to the lumbar puncture needle and the pressure of the C.S.F. is noted. The manometer is then removed and one or more specimens of C.S.F. collected in sterile tubes. Normal C.S.F. is water clear, and so it is in many diseases, such as tuberculous meningitis, disseminated sclerosis or general paralysis, in which the pathologist can find many abnormalities on testing the fluid. Cloudy or purulent fluid means that the meninges have been attacked by pus-forming organisms such as the meningococcus, as in meningitis. Blood-stained fluid may be due to bad technique, but C.S.F. consistently mixed with blood indicates a cerebral or subarachnoid hæmorrhage.

(5) Tapping the Chest

The chest may be needled to see if there is any fluid in the pleural cavity, to see what kind of fluid is present, or to drain the fluid away if it is harming the patient. Small quantities of non-purulent fluid do not generally need draining, but very large quantities of fluid compress the lung and push the heart out of place, and draining may then be necessary. Pus-containing fluid may have to be removed and antibiotics injected (see p. 98).

If the chest is to be needled simply to obtain a sample of fluid to send to the laboratory for diagnostic purposes all that need be laid out is a 5 or 10 ml. syringe, needles, local anæsthetic, some sterile swabs and towels. Sterile tubes should be ready for the collection of samples of fluid. The doctor will say where he intends to insert the needle; if the left side of the chest is to be approached, the patient lies on his right side with a pillow under his ribs and his left arm above his head. This position by separating the ribs as much as possible makes it easier to insert the needle between them. If the back of the chest is to be needled the patient sits up and leans forwards or lies curled up on his side.

If large quantities of fluid are to be drained a 50 ml. syringe and a wide-bore needle will be needed as well as a small syringe and needle for the local anæsthetic. A three-way connection between the needle and the syringe saves a lot of time, because the syringe can then be emptied without disconnecting it.

(6) Blood Transfusion*

It is often the duty of the nurse assisting at a blood transfusion to fetch the bottle of blood. This sounds simple, but it is a most responsible task. The messenger must be absolutely certain she has

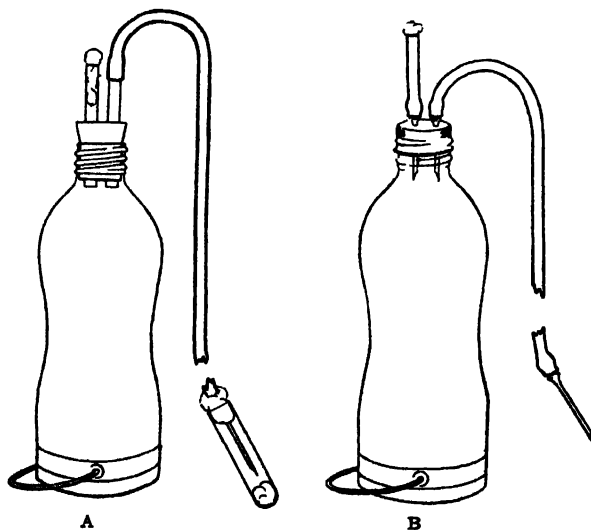


Fig 108 Blood Taking Set This set is used when blood is taken from a donor. The bottle contains sterile citrate solution to prevent clotting of blood for transfusion. If the blood is to be used for the preparation of serum a dry bottle is used. When the bottle is full the cork is removed and the bottle closed with a metal screw cap. The blood is then stored in the refrigerator. *N.B.*—This set can also be used for venesection, for instance in cases of hypertension.

* I am indebted to Dr. Richard Asher for many of these suggestions.

been given the blood for the right patient. Disasters have occurred when blood, labelled with the patient's surname only, has been given to a patient with the same name but different blood group. It is generally advised that each bottle should be labelled with the recipient's full name, ward and hospital number, as well as the blood group.

The standard transfusion set consists of (a) a bottle of blood with a screw cap, and an attached label stating the group of the blood, and (b) a tin box containing a complete set of rubber tubes and needle, ready sterilised and wrapped in cellophane. (It is possible that some transfusion reactions are caused by the rubber, so plastic tubes, and all-plastic giving sets, are being tried out.)

Put two transfusion sets (one as a spare) on a trolley, also sterile towels, swabs, antiseptic solution and two or three pieces of adhesive strapping (for fixing the needle after it has been inserted into a vein). The pieces of strapping should be put somewhere handy, such as attached to the head of the bed. Have a transfusion stand ready, look at the patient's arms, and arrange the one which seems to have the best veins on a pillow covered with a rubber sheet.

Now arrange a tourniquet around the patient's upper arm. It is best to use a sphygmomanometer with the tubes at the shoulder end, out of the way of the transfusion. Tighten the tourniquet or pump the sphygmomanometer up to 70 or 80 mm. so that the doctor can examine the veins; then lower the pressure until he is ready to insert the needle. Open the tin containing the tubing, etc., and hand it to the doctor. While he unwraps and disentangles it, show the bottle with its label, to the doctor, to make sure it is the right group, and remove the screw cap. The doctor will now insert the cork and tubing into the bottle. Hold the bottle against your hip while he rams the cork home—this avoids the disaster of the bottle's coming uncorked on turning it upside down and flooding the ward with blood. When the doctor has adjusted the screw clip so that it is, practically closed he will ask you to turn the bottle upside down, when blood should flow down the long tube and through the drip chamber. Leave the little cork in the air inlet tube, but ease it slightly if it gets jammed. When a few drops of blood have dripped out of the needle the screw clip is screwed tight; tighten the tourniquet again and all is ready for the insertion of the needle into the vein.

This may be a very difficult or quite an easy performance. When the needle is in the vein the doctor will unscrew the clip (after connecting up the needle if he has disconnected it) and blood will flow freely through the tubing and into the patient. Remove the

little cork from the air inlet tube, and undo the tourniquet, or disconnect the sphygmomanometer cuff or, of course, the blood will not flow. The clip is adjusted so that the blood flows or drips at the required rate. The needle is now fixed to the arm with strapping.

A well-padded splint may be necessary to immobilise the arm, but if the needle is inserted into a wrist or forearm vein, and if the patient is co-operative, immobilisation is often unnecessary.

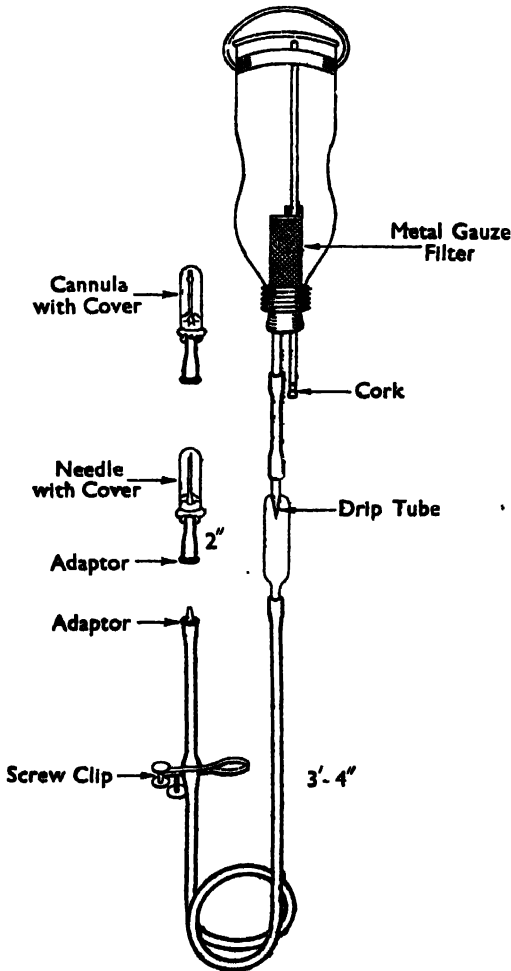


Fig. 109. Blood Transfusion Set. The diagram shows the set assembled and ready for use. The large cork carries two tubes, one to let blood out, the other to let air in. The air inlet tube is closed by a little cork which is removed when the transfusion starts running. The filter keeps back minute clots and other particles. (Sometimes a "gas mantle" filter is used instead of the metal one shown.) The glass drip tube shows how fast the transfusion is running. The adjustable screw clip regulates the flow. The needle (or cannula) is withdrawn from its glass cover just before its insertion into the vein; some doctors fit the needle on to the tubing before doing this, others wait until they are sure the needle is in the vein before connecting up

You will be told how fast the blood ought to run—do see that this rate is kept up, for the blood sometimes starts to run much faster or slower all of a sudden, when the screw clip will need adjusting. When several pints have to be given have the next bottle ready before the first one runs out; the bottles are changed by transferring the cork bearing the tubing from one to the other.

Inform the doctor *immediately* if the transfusion stops or if blood leaks out of the vein, raising a lump under the skin. Nothing is more maddening than to walk into the ward and find that a transfusion has stopped running, and that it is too late to do anything about it.

If the doctor cannot get into a vein with a needle he may have to cut down. This is generally done on an arm vein, and another trolley must be laid out with syringe, local anæsthetic, scalpel, forceps, artery forceps, aneurysm needle, catgut, cannula, needles, scissors and silkworm gut. A vein is exposed and the cannula tied in and connected up to the bottle of blood in the same way as a needle. The incision is protected with a sterile dressing, the limb kept still with sandbags or a splint. The nurse will be responsible for seeing that the rate of flow of blood is kept constant just as in the ordinary form of blood transfusion.

Transfusions and drips are not given into leg veins unless for some reason the arm veins cannot be used; this is because venous thrombosis is apt to develop in leg veins, with the possibility of complications such as leg ulcer or pulmonary embolism.

(7) Collecting Specimens for the Laboratory

Nurses often have to collect specimens for the laboratory, or to provide the doctor with the correct container for some specimen he is collecting. Here are a few of the specimens that often have to be sent to the laboratory, and the containers necessary for their collection.

Specimens of Blood for various tests generally have to be collected in sterile tubes. Sometimes they have to be kept from clotting, when a special chemical is added to the tube. In such cases mix by tilting the test tube; do not shake until the blood froths.

(a) *Blood Count*.—As a rule the blood counter brings his own apparatus. But if the blood has to be sent away to be counted a *fluoride* tube is necessary. The fluoride acts as an anti-clotting agent and preservative. About 1 ml. of blood is required.

(b) *Blood Sugar*.—Here, again, the pathologist may collect his

own specimen; otherwise a fluoride tube is used. One or two ml. are required.

(c) *Blood Sedimentation Rate*.—You will probably have to do this test yourself if you work in a T.B. or rheumatism ward. About 2 ml. of blood is collected in a tube and prevented from clotting by means of *citrate solution*. Suck the citrated blood up into a Westergren tube up to the 200 mm. mark and stand upright in the special stand. As the red blood corpuscles settle in the tube a clear layer of fluid appears at the top of the column of blood. Record how far down the tube the clear layer extends at the required intervals (say after one and two hours) thus:

	Mr. J.	B S.R.	May 6th.
<i>Time</i>	<i>Reading</i>		<i>Result</i>
11.45	0 mm.	B.S.R.	15 mm. in one hour and
12.45	15 mm.		25 in two hours.
1.45	25 mm.		

(d) *Blood Urea*.—About 5 ml. blood in a tube containing *oxalate* crystals (to prevent clotting) are required.

(e) *Blood Wassermann and Kahn reactions*.—Clotted blood is wanted for these tests, so use a clean dry tube. About 2 ml. of blood are wanted.

(f) *Widal reactions, etc.*—Here again clotted blood is wanted. About 5 ml. in a perfectly clean, dry tube.

(g) *Blood Culture*.—When we want to grow bacteria from the blood (say, in a case of bacterial endocarditis) we need to be extra careful that no other organisms get into the specimen, so scrupulous asepsis is necessary. The laboratory will probably provide sterile tubes containing the broth that is used to nourish the bacteria, and also a special syringe, ready dried and sterilised in a sterile tube. Ether is generally used to cleanse the skin, because it evaporates quickly and so does not get into the syringe, where it might damage the bacteria for which we are searching. The blood is squirted straight from the syringe into the tubes of broth.

2. *Urine*.—A *catheter specimen* is collected in a sterile, screw-topped bottle. Any container will do for a non-catheter specimen.

3. *Sputum*.—Get the patient to spit his early morning sputum into a screw-topped jar, and make sure he is spitting up sputum and not just saliva.

4. *Stools*.—A screw-topped jar is used. You may be asked to hurry the specimen to the laboratory, as some of the bowel organisms and parasites are delicate and difficult to grow.

5. *Cerebrospinal Fluid*.—A sterile, screw-topped tube is required (sometimes two or three, for different tests, and one containing citrate solution may be asked for). Hurry specimens of C.S.F. to the laboratory, as certain changes occur if the fluid is left standing about.

6. *Pus, pleural fluid*, etc. Sterile tubes are required; sometimes citrate solution is necessary, as some of these fluids clot like blood.

APPENDIX B

WEIGHTS AND MEASURES

Most confusingly, there are two systems of weights and measures in use in the prescribing of medicines; unfortunately it is essential to know them both. The *Imperial System* is the older of the two; in this country it is generally used when prescribing older remedies. The *Metric System* is the only one in use in most other countries; in Britain most of the newer drugs are prescribed in Metric weights and measures. Tables for converting doses from one system to the other are given below.

IMPERIAL WEIGHTS

60 grains (gr.) = 1 drachm (dr., ʒ)

8 drachms = 1 ounce (oz., ʒ) = 480 grains

METRIC WEIGHTS

1000 micrograms (μg) = 1 milligram.

1000 milligrams (mg.) = 1 gramme (g., G.)

1000 grammes = 1 kilogram (Kg.)

APPROXIMATE EQUIVALENTS: IMPERIAL TO METRIC WEIGHTS

grain	milligram	grain	gramme
1/100	0.6	1	0.0
1/50	1.2	5	0.3
1/10	6.0	10	0.6
1/8	8.0	15	1.0
1/6	10.0	20	1.3
1/4	16.0		
1/2	30.0	$\frac{1}{2}$ ounce = 15 gramm	

METRIC TO IMPERIAL WEIGHTS

10 milligrams = 1/6 grain

1 gramme = 15 grains

IMPERIAL MEASURES

60 minims (min., ℥) = 1 fluid drachm (dr., ℥)

8 drachms = 1 fluid ounce (oz., ℥)

20 fluid ounces = 1 pint

2 pints = 1 quart

4 quarts = 1 gallon

METRIC MEASURES

1000 millilitres (ml.) = 1 Litre — (for practical purposes)

1000 cubic centimetres (c.c.)

APPROXIMATE EQUIVALENTS: IMPERIAL TO METRIC MEASURES

nims	Millilitres
1	0.06
2	0.12
5	0.3
10	0.6
15	1.0
60	4.0

1 fluid ounce = 30 millilitres

1 pint = 568 millilitres

METRIC TO IMPERIAL MEASURES

1 millilitre = 1 c.c. = 15 minims

1 litre = 35 ounces = $1\frac{3}{4}$ pints

PHARMACEUTICAL LATIN TERMS AND ABBREVIATIONS

The Latin terms and their abbreviations, widely used in writing prescriptions in this country, are relics of the days when Latin was the universal language of all scholarly professions. The most recent edition of the British Pharmacopœia has finally abandoned the use of all Latin terms, but they are unlikely to die out altogether for many years to come.

LATIN	ABBREVIATION	ENGLISH
ana (Greek)	āā	of each
aqua	aq.	water
bis in die	b.i.d.	twice daily
compositus	co.	compound
cum	ċ	with
et		and
Guttae	Gutt.	drops
Haustus	Haust.	a draught
Linamentum	Lin.	liniment
Liquor	Liq.	solution
Lotio	Lot.	lotion
Mane		morning
Mistura	Mist.	mixture
mitte		send
omni nocte	o.n.	each night
Pilula	Pil.	pill
Pulvis	Pulv.	powder
post cibos	p.c.	after eating
pro re nata	p.r.n.	whenever necessary
quantum sufficiat	q.s.	as much as required
Recipe	℞	take
repetatur	rep.	let it be repeated
si opus sit	s.o.s.	if required
semis	ss	half
ter die sumendum	t.d.s.	to be taken thrice daily
Unguentum	ung	ointment

GLOSSARY

Introductory Note.—Nurses will find medical terms much easier to understand if they know the meanings of some of the common beginnings and endings of words, such as:

<i>A-, an-</i>	..	Lack of; e.g. anæmia, lack of blood.
<i>-æmia</i>	..	Blood; e.g. leukæmia, white blood.
<i>-cyte</i>	..	Cell; e.g. leucocyte, white cell.
<i>-ectomy</i>	..	Removal of; e.g. gastrectomy, removal of the stomach.
<i>hyper-</i>	..	Above, too much; e.g. hypertension, high blood pressure.
<i>hypo-</i>	..	Below, too little; e.g. hypoglycæmia, too little glucose in the blood.
<i>-itis</i>	..	Inflammation of; e.g. tonsillitis, inflammation of the tonsil.
<i>macro-</i>	..	Large; e.g. macrocyte, large cell.
<i>micro-</i>	..	Small; e.g. microcyte, small cell.
<i>-oma</i>	..	Tumour; e.g. adenoma, tumour arising from a gland.
<i>-osis</i>	..	Condition of, disturbed condition of; e.g. psychosis, disturbed condition of the mind.
<i>poly-</i>	..	Many, much; e.g. polyuria, copious urine.
<i>tachy-</i>	..	Rapid; e.g. tachycardia, rapid heart action.
<i>-uria</i>	..	In or of the urine; e.g. albuminuria, albumin in the urine.

A

Acetone.—Chemical formed in excess during abnormal breakdown of fat (e.g. in diabetes). Also found in nail varnish remover and lighter fuel.

Achalasia.—Failure to relax.

Achlorhydria.—Absence of hydrochloric acid in the stomach.

Adenoma.—Tumour arising from a gland.

Adrenal gland.—Ductless gland situated above the kidney.

Adrenaline.—Secretion of the adrenal gland.

Agranulocytosis.—Lack of white blood cells.

Albumin.—A protein found in normal blood and abnormal urine.

Albuminuria.—Albumin in the urine.

Allergy.—Abnormal sensitivity to various substances.

Alveolus.—Minute air sac (of lung).

Ankylosis.—Fixation of joint due to disease.

Aneurysm.—Dilatation of an artery due to weakness of its wall.

Anticoagulants.—Drugs which lessen the blood's clotting power.

Antitoxin.—Substance which antagonises bacterial toxin.

Anus.—Back passage.

Aorta.—Main arterial trunk leading out of left ventricle of heart.

Aperient.—Drug tending to loosen the bowels.

Aphasia.—State in which power of speech is wholly or partly lost, without any paralysis of tongue or lips.

Apoplexy.—Stroke.

- Arachnoid membrane*.—One of the membranes covering the brain and spinal cord.
- Atheroma*.—Disease of arteries in which plaques of fatty material are found in the vessel lining.
- Arthritis*.—Inflammation of joint.
- Arthrodesis*.—Fixation of a joint by operation.
- Ascites*.—Collection of fluid in abdominal cavity.
- Aspiration*.—Suction.
- Auditory*.—Connected with the ear.
- Auricles*.—The smaller chambers of the heart.
- Axilla*.—Armpit.

B

- Bacteria*.—Microscopic organisms, many of which cause disease.
- ✓ *Basal Metabolic Rate*.—Rate of consumption of energy of person at rest.
- Belch*.—Bring up wind.
- Biopsy*.—Removal and examination of tissue from living body for purposes of diagnosis.
- Bronchiectasis*.—Disease in which bronchi are dilated.
- Bronchitis*.—Inflammation of the bronchi.
- ✓ *Bronchopneumonia*.—Pneumonia in which patches of inflammation surround branches of bronchi.
- ✓ *Bronchoscope*.—Instrument for inspecting the bronchi.
- Bronchus*.—Tube leading from windpipe to lung.
- Bundle of His*.—Special strand of tissue connecting auricles and ventricles.

C

- Calorie*.—Unit used in the measurement of energy value of food. (Equivalent to amount of heat required to raise the temperature of one litre of water through one degree centigrade.)
- Capillaries*.—The smallest blood vessels.
- Carbohydrates*.—Group of food substances including starch and sugar.
- Carbuncle*.—Acute inflammation and pus formation under the skin.
- ✓ *Carcinoma*.—Cancer.
- ✓ *Cardia*.—Sphincter between œsophagus and stomach.
- Cardiac*.—Connected with the heart.
- Cardiac Asthma*.—Acute attacks of breathlessness, like those of asthma, occurring in heart failure.
- Carrier*.—Person who carries disease germs and is capable of infecting others, though not himself suffering from the disease.
- Carditis*.—Inflammation of the heart.
- Caseation*.—Formation of cheesy matter (e.g. in the lung).
- Cataract*.—Disease of the eye in which the lens becomes opaque.
- Cellulitis*.—Inflammation of connective tissue.
- Cerebral*.—Connected with the brain.
- Cerebrospinal fluid*.—Fluid which bathes the brain and spinal cord.
- Cheirpompholyx*.—Skin condition in which hands are covered with an itching rash consisting of sago-like spots.
- Cholecystitis*.—Inflammation of the gall bladder.

Cholecystogram.—X-ray of gall bladder.

Chorea.—St. Vitus Dance.

Cirrhosis.—Disease in which liver is hardened, irregular and knobbly.

Colon.—Part of large bowel.

Colitis.—Inflammation of colon.

Coma.—Unconsciousness from which the person cannot be awakened by ordinary methods.

Comedo.—Blackhead.

Congenital.—Inborn.

Congested.—Overfilled (e.g. with blood).

Congestive Heart Failure.—Heart failure in which the veins of the lungs or body generally are overfilled with blood.

Consolidation.—Solidification (of part of lung in pneumonia).

Consumption.—Tuberculosis of the lungs.

Contracture.—Deformity of joint due to abnormal contraction of muscle.

Convulsions.—Fits.

Coronary arteries.—Arteries supplying the heart with blood.

Corpuscles.—Cells (of blood). Literally, minute bodies.

D

Defæcate.—Have bowels open.

Dehydration.—Loss of water.

Dermatitis.—Inflammation of skin.

Diagnosis.—To make a diagnosis is to decide from the signs and symptoms what disease is present.

Diaphragm.—Muscle separating chest from abdomen.

Diarrhæa.—The frequent passage of loose stools.

Diastole.—Period of relaxation of heart between beats.

Diathesis.—Constitution, tendency (to disease).

Diuretic.—Tending to promote the flow of urine; drug which does this.

Disseminated.—Scattered.

Dropsy.—Collection of fluid in tissues.

Duodenum.—Part of small intestine leading out of stomach.

Dysentery.—Inflammation of the bowel, caused by bacteria or other parasites, resulting in diarrhæa.

Dyspepsia.—Indigestion.

Dysphagia.—Difficulty in swallowing.

Dyspnœa.—Breathlessness.

E

Effusion.—Collection of fluid, e.g. in pleural cavity.

Electrocardiogram.—Record of electrical currents in heart.

Electrolytes.—Substances which in solutions will conduct electricity. (Electrolytes of importance in clinical medicine are certain salts—e.g. those of potassium and sodium—which are present in the blood and other body fluids.)

Embolism.—Blocking of an artery by foreign body (e.g. blood clot) in blood stream.

- Emphysema*.—Disease in which air sacs of lungs are overdistended with air.
- Empyema*.—Collection of pus in a body cavity (generally applied to collection of pus in pleural cavity).
- Encephalitis*.—Inflammation of the brain.
- Encephalomyelitis*.—Inflammation of the brain and spinal cord.
- Endemic disease*.—One found frequently in a certain district.
- Endocrine glands*.—Ductless glands—those which pass their secretions straight into blood stream.
- Endocarditis*.—Inflammation of endocardium.
- Endocardium*.—Lining of the heart.
- Enteritis*.—Inflammation of intestines.
- Epidemic*.—Outbreak (of infectious disease).
- Epistaxis*.—Nosebleeding.
- Erythema*.—Redness (of the skin).
- Exophthalmos*.—Bulging eyes.
- Expectorant*.—Drug which helps to increase the flow of sputum.
- Exudate*.—Fluid produced as a result of inflammation.

F

- Fæces*.—Stools, contents of lower bowel.
- Febrile*.—Feverish.
- Fever*.—Condition in which patient's temperature is raised—i.e. the interior of the body is hotter than normal.
- Flatulence*.—Wind, belching.
- Fœtus*.—Infant in womb.
- Fracture*.—Break (of bone).

G

- Gallstones*.—Stones which form in the gall bladder.
- Ganglion*.—Group of nerve cells.
- Gangrene*.—Death of a large part of a limb or organ, generally caused by cutting off its blood supply.
- Gastrectomy*.—Removal of stomach.
- Gastric*.—Connected with the stomach.
- Gastritis*.—Inflammation of the stomach.
- Gastroenteritis*.—Inflammation of the stomach and intestines.
- Gastrostomy*.—Artificial opening between stomach and exterior.
- Glomerulus*.—Special tuft of minute blood vessels in kidney.
- Glucose*.—A kind of sugar, present in blood and tissues.
- Glycogen*.—A starchy substance normally stored in the liver and muscles.
- Glycosuria*.—Glucose in the urine.
- Goitre*.—Enlargement of the thyroid gland.

H

- Hæmatemesis*.—Vomiting blood.
- Hæmoglobin*.—Red colouring matter of blood.

Hæmolysis.—Breakdown of red cells which liberates hæmoglobin.

Hæmoptysis.—Blood spitting.

Hæmorrhage.—Bleeding.

Hemiplegia.—Paralysis of one side of the body

Hepatic.—Connected with the liver.

Hepatitis.—Inflammation of the liver.

Homologous.—Corresponding (Homologous serum jaundice = jaundice caused by serum from same species, i.e. man).

Hormones.—Chemical substances produced by ductless glands.

Hydrochloric acid.—Acid normally present in the stomach.

Hyperglycæmia.—Too much glucose in the blood.

Hyperpyrexia.—Very high fever.

Hypertension.—High blood pressure.

Hypertrophic.—Overgrown.

Hypnotic.—Sleep compelling; sleep compelling drug (e.g. barbitone).

Hypochromic.—Too little colour.

Hypoglycæmia.—Too little glucose in the blood.

Hypostatic pneumonia.—Inflammation of lowest parts of lungs (e.g. back of lungs of patient lying on his back).

I

Idiopathic.—Of unknown cause. (Literally - self-originated).

Ileum.—Lower part of small intestine.

Ileus.—Obstruction of the intestines.

Ileostomy.—Artificial opening into ileum.

Incontinent.—Unable to control (urine, fæces, etc.).

Incubation period.—Period elapsing between infection and appearance of symptoms of disease.

Infarct.—Part of organ (e.g. heart) whose blood supply has been cut off.

Infection.—Invasion by disease-producing organisms.

Insomnia.—Sleeplessness.

Intercostal.—Between the ribs.

Interstitial tissue.—Packing tissue.

Intracranial.—Inside the skull.

J

Jaundice.—Yellowness due to excess bile pigments in blood.

†

K

Ketones.—Chemicals, such as acetone, formed in excess during abnormal breakdown of fat in the body (e.g. in diabetes).

Ketosis.—Accumulation of ketones in body.

L

Lactation.—Secretion of milk.

Laxative.—Tending to loosen the bowels; drug which does this.

Laryngitis.—Inflammation of the larynx.

Larynx.—Voice box.

Larva.—Early stage in the development of an insect.

Leucocytosis.—Increase in the number of white blood cells.

Linctus.—Sweet mixture intended to stop coughing.

Lobar pneumonia.—Inflammation of lobe of lung.

Lobectomy.—Removal of lobe (e.g. of lung).

M

Macrocytic.—Large-celled.

Meconium.—Dark slimy material present in intestines of new-born baby.

Melæna.—Passage of stools which are black because they contain altered blood.

Meninges.—Membranes covering the brain and spinal cord.

Meningitis.—Inflammation of the meninges.

Menorrhagia.—Heavy menstrual periods.

Metabolism.—Sum of chemical processes going on in body.

Miliary.—The size of millet seeds (bird seed).

Miliary Tuberculosis.—Tuberculous septicæmia in which miliary patches of tuberculous tissue are scattered throughout the body.

Microcytic.—Small-celled.

Micturition.—Passing urine.

Mitral valve.—Valve between left auricle and ventricle (so called because it has two flaps, and hence resembles a bishop's mitre).

Motor nerves.—Nerves which control movements of muscles.

N

Necrosis.—Death of tissue.

Nephritis.—Inflammation of kidney.

Nerves.—Thread- or cord-like structures which convey impulses to and from the brain and spinal cord; not to be confused with

"*Nerves*."—Emotional instability.

Neuralgia.—Pain along the course of a nerve.

Neuritis.—Inflammation of nerve.

Neurosyphilis.—Syphilis of the nervous system.

Nits.—Louse eggs.

Night blindness.—Inability to see in the dark.

Nystagmus.—Oscillation of eyeballs.

O

Occlusion.—Blockage.

Occupational Therapy.—Treatment of patient by giving him something to do.

Edema.—Dropsy; collection of fluid in tissues.

Œsophagus.—Gullet.

Optic nerve.—The nerve of the eye on which vision depends.

Ophthalmoscope.—Instrument for examination of interior of eye.

Oral.—By mouth.

Orchitis.—Inflammation of the testicle.

Orthopnoea.—Inability to breathe adequately except when sitting upright.

Otitis.—Inflammation of the ear.

Otitis media.—Inflammation of the middle ear (just inside the ear drum).

P

Palpitation.—Consciousness of heart beat.

Palsy.—Paralysis.

Pancreas.—A gland which sends digestive juice into the duodenum and insulin into the blood.

Papilloedema.—Swelling of the optic nerve.

Paracentesis.—Drainage of fluid (e.g. from abdomen).

Parasite.—Plant or animal which feeds on living plant or animal.

Paralysis.—Inability to move.

Parotids.—The largest of the salivary glands.

Parotitis.—Inflammation of parotids.

Pasteurisation.—Heat treatment of milk intended to kill disease-producing germs without boiling.

Pediculosis.—Infestation with lice.

Pelvis.—Outlet (e.g. of kidney.)

Peptic ulcer.—Ulcer of stomach or certain adjoining organs.

Pericarditis.—Inflammation of pericardium.

Pericardium.—Membrane covering heart.

Peripheral.—At the outskirts.

Peripheral neuritis.—Inflammation of ends of nerves.

Peristalsis.—Wave-like contractions, e.g. of gut, which urge its contents along.

Peritoneum.—Membrane lining abdomen and covering abdominal organs.

Peritonitis.—Inflammation of peritoneum.

Phrenic.—Connected with the diaphragm.

Physiotherapy.—Treatment by physical means—e.g. massage, electricity, etc.

Pituitary.—Ductless gland situated in the skull below the brain.

Plasma.—Fluid part of blood.

Platelets.—Minute bodies in blood, concerned with blood clotting.

Pleura.—Membrane covering lungs and lining chest.

Pleurisy.—Inflammation of pleura.

Pleurodynia.—Pain in chest resembling that of pleurisy.

Pneumococcus.—Organism usually responsible for lobar pneumonia.

Pneumectomy.—Removal of lung.

Pneumonia.—Inflammation of lung.

Pneumoperitoneum.—Air in the peritoneal cavity.

Pneumothorax.—Air in the pleural cavity.

Polyarthritis.—Inflammation of several joints.

Polycythæmia.—Excess of red blood cells.

Polymorphs.—White blood cells which have the power of engulfing bacteria, etc.

Polyp.—Small outgrowth of mucous membrane.

Polyuria.—Passage of large quantities of urine.

Prognosis.—The outlook for the patient.

Prophyllaxis.—Prevention (of disease).

Protein.—Substance present in all living cells; essential food constituent.

Psychiatry.—The study of mental illness.

Pulmonary.—Connected with the lung.

Purge.—Drug tending to empty the bowel.

Purpura.—Rash consisting of purple spots and stains caused by hæmorrhages under the skin.

Purulent.—Consisting of or containing pus.

Pus.—Semi-liquid debris of dead tissue, white blood cells, etc., produced in certain types of inflammation.

Pyelitis.—Inflammation of outlet of kidney.

Pyelonephritis.—Pyelitis extending to kidney itself.

Pylorus.—Part of stomach opening into duodenum.

Pyramidal tract.—Nerve pathway connecting brain with motor cells of spinal cord.

Pyrexia. Fever.

Q

Quarantine.—Period during which contacts of infectious disease are isolated.

Quinsy.—Abscess around the tonsil.

R

Rectum.—Lowest part of large bowel.

Reflex.—Automatic action.

Regurgitation.—Backward flow.

Renal.—Connected with the kidney.

Respiration.—Breathing.

Retention.—Keeping back (of urine).

Retina.—Nervous lining of eye on which vision depends.

Retinitis.—Inflammation of retina.

Rigor.—Attack of shivering accompanied by rapid rise of temperature.

Rinorrhœa.—Running nose.

Roughage.—Indigestible residue of food.

S

Sciatic nerve.—Large nerve running down back of thigh and leg.

Sciatica.—Pain along course of sciatic nerve.

Sebaceous glands.—Glands which produce the natural grease of the skin.

Secretion.—The product of a gland.

Sedative.—Quietening; a quietening drug, e.g. phenobarbitone.

Septicæmia.—Infection in the blood stream.

Serum.—Fluid which separates when blood clots.

Shock.—Condition of collapse associated with pallor, rapid pulse, low blood pressure, etc.

- Sigmoidoscope*.—Instrument for examining rectum and lower part of colon.
- Sinusitis*.—Inflammation of nasal sinuses—certain cavities opening into the nose.
- Slough*.—Dead tissue which separates from ulcer.
- Sphygmomanometer*.—Apparatus for taking blood pressure.
- Spirochæte*.—Spiral organism, such as that causing syphilis.
- Splenectomy*.—Removal of spleen.
- Staphylococcus*.—Bacterium which often causes boils, carbuncles and other more serious infections.
- Status asthmaticus*.—Very prolonged attack of asthma.
- Status epilepticus*.—Repeated epileptic fits without return of consciousness between convulsions.
- Steatorrhoea*.—Condition in which fatty stools are passed.
- Stenosis*.—Narrowing (e.g. of heart valve).
- Sternum*.—Breastbone.
- Steroids*.—A group of substances including cortisone and its allies.
- Stomatitis*.—Inflammation of the mouth.
- Streptococcus*.—Bacterium responsible for scarlet fever, erysipelas and many other infections.
- Stupor*.—Semi-consciousness from which patient can be roused with difficulty.
- Subnormal*.—Below normal.
- Suppuration*.—Pus formation.
- Suprarenal gland*.—Ductless gland situated above the kidney (another name for *adrenal gland*).
- Sympathectomy*.—Surgical removal of sympathetic nerves and ganglia.
- Symptom*.—Something the patient complains of.
- Systole*.—Contraction (of heart).

T

- Tachycardia*.—Rapid beating of heart.
- Therapy*.—Treatment.
- Thrombosis*.—Clotting of blood in heart or vessels.
- Thrombus*.—Blood clot formed during thrombosis.
- Thoracoplasty*.—Operation in which part of chest wall is removed.
- Thoracoscope*.—Instrument for examination of pleural cavity.
- Thyroid*.—Ductless gland situated below Adam's apple.
- Thyroidectomy*.—Removal of thyroid gland.
- Thyrototoxicosis*.—Disease in which the thyroid gland is overactive.
- Thyroxine*.—Chemical substance produced by thyroid gland.
- Tonsillitis*.—Inflammation of tonsils.
- Toxæmia*.—Illness due to toxins in the blood stream.
- Toxic*.—Poisonous.
- Toxin*.—Poison: usually applied to poisons produced by bacteria.
- Trachea*.—Windpipe.
- Tracheotomy*.—Opening into the trachea.
- Tumour*.—New growth; swelling.

U

Uræmia.—Set of symptoms caused by the accumulation in the blood of waste products normally got rid of in the urine.

Urea.—Chemical formed during the breakdown of protein in the body and normally discharged in urine.

Uric acid.—Another chemical normally discharged in urine.

Uterus.—Womb.

V

Vaccination.—Inoculation with cowpox virus (*vaccinia*) to induce immunity to smallpox. Other forms of prophylactic inoculation.

Vaccine.—Preparation (usually of dead bacteria) used to increase immunity of subject to infection with similar organisms.

Vagina.—Passage leading from uterus to exterior.

Varicose veins.—Dilated irregular veins.

Venesection.—Blood-letting; opening of vein for removal of blood.

Ventricles.—The two larger chambers of the heart; the cavities in the brain.

Vesicle.—Minute blister.

Virus.—Organism smaller than bacteria; too small to be seen with microscope.

Vitamin.—Substance present in minute quantities in food, which is necessary to health.

W

Wassermann Reaction.—A blood test for syphilis.

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